EFFECTS OF RACTOPAMINE HCl ON PHYSICAL AND REPRODUCTIVE PARAMETERS IN THE HORSE

A Thesis

by

RUSSELL DEREK KRIEWALD

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

May 2008

Major Subject: Animal Science

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Approved by:

Chair of Committee,	Martha M. Vogelsang
Committee Members,	Brett D. Scott
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ABSTRACT

Effects of Ractopamine HCl on Physical and Reproductive Parameters in the Horse. (May 2008)

Russell Derek Kriewald, B.S., Texas A&M University

Chair of Advisory Committee: Dr. Martha M. Vogelsang

The objective of this study was to monitor the effects on physical and reproductive parameters in mares supplemented with Ractopamine HCl (RAC), in an effort to provide some insight concerning the use of RAC in horse diets. Physical deviation was recorded via measurements of body weight (BW), muscle size, and fat deposition. Reproductive deviation was recorded via ultrasonographic measurement of follicular growth and ovulation, while hormonal analyses were conducted for leptin and luteinizing hormone (LH). Data analyses of physical measurements indicated an effect of RAC supplementation ($P<.001$) as treated horses had a greater increase in BW compared to the controls. Treatment horses increased gaskin circumference ($P<.001$) compared to horses on the control diet. Both groups showed an increase in rump fat with the treated horses gaining less ($P<.05$). A similar effect was revealed in body fat percentage ($P<.01$) with the treated horses gaining less when compared to the controls. No statistical differences were noted for changes in forearm circumference or rib fat. No change was derived for length of estrous or pre-ovulatory follicle size between groups. When analyzing the data from first to last cycle in the treatment group, length of estrous was significantly ($P<.05$) shortened over the 90-d study. Upon analysis of serum leptin concentrations, the control group had a significantly ($P<.001$) higher overall

concentration as compared to the treated horses; however, no difference was noted for normalized data, though RAC supplementation may have caused the profile of leptin to become more erratic. Analysis of LH concentrations revealed a strong trend (P=0.0527) of RAC-supplemented horses having a lower mean concentration of LH throughout the 90-d study as compared to the controls. Means were also analyzed for day and treatment by day effects, suggesting possible trends (P=0.2944 and P=0.1591 respectively) of seasonality. Area Under the Curve (AUC) was calculated for individual horses and analyzed for treatment effects. Only a trend (P=0.1631) was noted for RAC-supplemented horses having a smaller AUC (80.10 ± 29.72) as compared to the controls (140.60 ± 27.50).

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May God Bless you all and provide you with the strength and will to achieve whatever it is you desire!

"*What lies behind us and what lies before us are but tiny matters compared to what lies within us.*" - R.W. Emerson

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CHAPTER I

INTRODUCTION

Today's horse industry is an extremely competitive market that is continuously evolving. Over time, breeders have manipulated and produced bloodlines that began with a strong foundation of quality breeding stock. To be productive, a breeder must choose individual animals for breeding based on quality, conformation, physical ability, and temperament. However, owners and trainers oftentimes fail to consider the effects on other parameters of physiology that a certain product or practice can cause. It is this train of thought that necessitates extensive research into any product which is used to enhance show ring preparations and performance of the horse.

A mare intended for breeding must be in optimal body condition to efficiently achieve and maintain a pregnancy to term. Nevertheless, for a show ring appearance, mares are expected to be more fit, representing the ideal standards for the particular class. At times, owners will go to any extent to gain a competitive edge in the show ring. One way of achieving this edge is by introducing the use of a supplemental product that gives the desired physical changes in the horse's training program. While use of such products can give desirable outcomes in the near term, they may decrease the mare's worth as breeding stock after her show ring career.

There are numerous products that claim to enhance the performance of the horse, thus giving the competitive edge a horseman might desire. These enhancements could include lean muscle growth resulting in muscle strength, delay of fatigue, overall growth,

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This thesis follows the style and format of the Journal of Animal Science.

or any other effect which could better prepare a horse for the intended task. One of the most recognized products used to obtain these effects is an anabolic steroid. Though these steroids can have a therapeutic use for debilitated horses, they also present the possibility to enhance a horse's maximal athletic performance. After research identified adverse effects to both the mare's and stallion's reproductive physiology and behavior, steroid usage in competitive horses was banned throughout the industry. Now, horsemen seek the next available "miracle" product to obtain the same effects. Many times, species barriers are crossed, and uneducated decisions are made which jeopardizes the overall reproductive success in horses.

It has been suggested that Paylean®, a feed additive developed for swine, is currently being used to enhance performance in the horse industry. Ractopamine HCl (RAC) is a β-adrenergic agonist and growth regulator that is marketed under the product names Paylean® (for swine) and Optaflexx® (for cattle) (Elanco, Indianapolis, IN). Diets supplemented with RAC as an active ingredient have been shown to promote lean muscle gain and feed efficiency in swine via stimulation of lypolysis and protein synthesis, as well as the down regulation of lipogenesis (Kelly et al., 2003). Due to steroid-like effects and the depletion of adipocytes, there should be concern with respect to effects on reproductive physiology.

In swine, Paylean® is not recommended for use in breeding gilts. The dramatic decrease in adipose tissue could potentially have severe negative effects on the animal's hormone profile and its ability to remain reproductively sound. Leptin, which is suggested to be the hormonal factor that signals the nutritional status to the reproductive system, is secreted in a circadian rhythm by adipocytes and is therefore likely to mediate

growth and reproductive performance. When adipocytes are depleted, there are fewer cells capable of secreting leptin. As a lack of leptin suppresses hypothalamus and pituitary function, gonadatrophic hormones such as luteinizing hormone (LH) and follicle stimulating hormone (FSH) are likely to be inhibited, thus suppressing reproductive performance by interfering with normal ovarian function.

The results of this study could introduce key knowledge on how dietary supplementation of horses with Paylean® affects physical and reproductive parameters in the horse. Members of the horse industry may find this information applicable to the current off-label administration of Paylean® in horses, and as well, may find it pertinent to strictly regulate the use of the product. It is critical to uncover any possible routes in which a toxicity or disruption in physiology may occur in order to prevent such detriments to the industry.

CHAPTER II

REVIEW OF LITERATURE

β-agonists – An Overview

Nearly every mammalian cell type contains β-adrenergic receptors (β-AR) that are stimulated via norepinephrine and epinephrine. The physiological response of particular cells depends upon the composition of the β-AR subtypes present, and as well the species-specific amino acid sequence. Oral administration of some β-agonists can cause accretion of skeletal muscle and decreased fat deposition in swine, cattle, and other meat-animal species. Knowledge of those effects proposes direct actions on both skeletal muscle and adipose tissue. However, the overall effect of a given β-AR within a given species may also be controlled by other physiologic mechanisms including blood flow, hormone secretion, and by feed intake as controlled by the central nervous system (Mersmann, 1998). As animals mature and grow, not only do they receive differing diets, but cells change and proliferate, which may cause the animal to become more or less sensitive to such a product. Additionally, as species differ in feed consumption and hormonal profiles, the effects of such β-agonists would likely differ.

Increased skeletal muscle growth is the primary effect noted when a β-agonist is administered to a meat-animal species. It is speculated that the cause of increased muscle growth is due to an increase in protein synthesis, a decrease in protein degradation, or a combination of both (Yang and McElligott, 1989; Moloney et al., 1991). Though these mechanisms have been demonstrated in previous research, some reports have been unable to demonstrate such actions (Bergen et al., 1989; Claeys et al., 1989).

Secondary to muscle growth is the depletion of adipose tissue, or a decrease in carcass fat mass due to stimulation of adipocyte degradation. The effects on adipose tissue, however, are not as consistent as the finding on skeletal muscle. Though a decrease in fat mass is reported when such an agonist is fed, some studies have indicated little or no effects on lipid metabolism in-vitro (Spurlock et al., 1993, 1994).

Due to the presence of β-AR on multiple cell types throughout the body, the effects of a β-agonist are likely to be more extensive, as well indirect, in the recipient animal. β-agonists, such as epinephrine, increase blood flow to various parts of the body. For instance, increasing blood flow to skeletal muscle tissue can certainly increase the amount of substrates and energy available for protein synthesis and muscle growth. Additionally, an increased blood flow through adipose tissue can assist in transporting nonesterified fatty acids away from the tissue, thus enhancing the rate of lipid degradation (Mersmann, 1998). Increased blood flow to the hindlimbs has been noted for both cattle (Eisemann et al., 1988) and sheep (Beermann et al., 1987), and both skeletal muscle and adipose tissue in swine (Mersmann, 1989). These effects are likely a result of the increase in heart rate noted for multiple β-agonists (Mersmann, 1987), and may further assist the more direct and commonly noted effects in meat-animal species.

Ractopamine HCl (RAC)

Ractopamine HCl (RAC) is a β-adrenoceptor agonist that exists in two diasteriomeric forms due to the presence of two chiral carbons. The racemic preparation has been approved as a feed additive for use in finishing swine and cattle. Ractopamine HCl is utilized as a growth promotant, and has a repartitioning effect by transferring

nutrients from fat deposition to protein synthesis and accelerated lean muscle gain. Developed by the Elanco Animal Health company, RAC was first approved by the FDA for use in finishing swine in 2000 to facilitate growth and feed efficiency. To stay competitive with other meat species, profit driven swine producers utilize RAC, in the commercial formulation of Paylean®, to provide health-conscious consumers with a leaner meat product.

Previously, RAC has been found to be absorbed in the gastrointestinal tract when supplemented orally, and was detected by metabolites found in horse urine (Lehner et al., 2004). The same study noted that, due to the metabolites recovered, RAC is metabolized in a similar way as in other species, such as swine, even though the rate of supplementation was considerably lower (less than 1mg/kg BW fed to horses compared to a range of 5-20mg/kg BW fed to pigs). Buff et al. (2006) hypothesized the potential efficacy of using RAC as a weight loss supplement in obese pony mares in a negative energy balance. Comparisons were made when ponies were fed RAC at 0 mg/kg, 0.6 mg/kg, and 1.0 mg/kg BW. All pony mares, regardless of dietary group, decreased body weight over time. However, a greater decrease in BW was observed when RAC was fed at 1.0 mg/kg as compared to the control (0 RAC) and 0.6 mg/kg groups. This decrease in body weight could be further enhanced by the fact that all ponies were restricted to 75% of ad libitum intake. Thus, it was possible to use RAC in order to decrease BW in obese ponies. This finding is especially important due to the fact that obesity is commonly associated with other problems, such as laminitis, that inhibit the horse's ability to exercise adequately. Some horses are genetically predisposed to obesity. In swine, research has proven RAC can be utilized to improve the efficiency of feed utilization

while promoting the leanness of a carcass regardless of the propensity of fat deposition (Yen et al., 1990). Swine supplemented with RAC had less fat depth, yet a higher dressing percentage was achieved.

Prior studies in swine commonly note that consumption of RAC at differing levels resulted in increases in BW and percent yield and lean. Kelly et al. (2003) evaluated the response of feeding RAC on weight gain, feed intake, carcass value and economic return in finishing pigs. This study demonstrated an increase in body weight over the 4-wk feeding period, with 50% of the total weight gain occurring within the first week of treatment. Additionally, percent yield and lean also increased over the duration of the study. Though horses are not typically recognized as market animals nor evaluated for carcass characteristics, the goal of lean muscle growth remains the same, especially for show ring appearances.

Toxicological Effects

As new products are introduced to alter an animal's physiologic state, creating toxicity within that individual should be of concern. This is especially true when products are fed off-label, such as in a non-approved species, and the consumer lacks the appropriate information to make sound decisions. The use of RAC in horse diets is not approved, and is therefore illegal. Little evidence is available to judge the safety of RAC in horses. Research to date (Buff et al., 2006), including the present study, has fed relatively low levels of RAC compared to the typical dose administered to swine. The upper limits of RAC supplementation in horse diets is unknown, therefore the issue

should be approached cautiously. Though β-agonists have varying effects across species, information can be gathered from other species to make a hypothesis.

Unfortunately, most of the toxicological data obtained by Williams and associates has been conducted through the Toxicology Division of Lilly Research Laboratories (Elanco Animal Health, Indianapolis, IN), and is unpublished. Initially, short-term studies in toxicology were conducted on crossbred swine. Though swine tolerated the highest does of 15mg/kg BW per day without physical signs of toxicity, erythrocyte number and volume fraction, as well as hemoglobin concentrations were decreased. However, a no-observed-effect level (NOEL) was not identified (Williams et al., 1987 as found in Ungemach, 2004). In a long-term study conducted on both male and female mice, it was found that the highest dose of RAC for both males (840 mg/kg BW per day) and females (1085mg/kg BW per day) exceeded the maximum tolerance level for mice (Williams, 1998 as found in Ungemach, 2004). The study noted a 25% survival rate for males and a 32% survival rate for females at the highest dose. Mortality rates at this dose were thought to be attributed to an increased severity of cardiomyopathy. For the male mice, the NOEL was determined to be 25mg/kg BW per day. However, a NOEL was not established for the female as a dose-dependant increase of uterine leiomyomas.

A Comparison to Anabolic Steroids

Anabolic steroid use in the equine industry has been of great interest for several decades due to its ability to alter or improve performance. Since then, steroid usage has been closely monitored and restricted by all equine organizations, though many horse owners still insist on finding a substitute for steroids without considering adverse effects.

The comparison between RAC and steroids is an important issue as both products can induce similar physical changes including an increase in BW and lean, delineated muscle gain. However, they might not cause the same changes from a reproductive aspect as RAC is instead, a growth promotant, not a hormone. A study conducted in 1980 analyzed the effects on reproductive function of two different steroids in yearling mares (Maher et al., 1983). Investigators noted that all mares receiving an anabolic steroid treatment failed to display normal estrous behavior; instead a more aggressive behavior toward other horses was noted. Additionally, the steroid treatment resulted in mares having fewer estrous cycles and ovulations, as well as a decrease in ovarian size and number of growing follicles. These occurrences can be further supported by the depression of LH, FSH, and progesterone that was observed. Disruption of the normal hormonal profile would essentially cause a negative effect on reproductive performance by suppressing the normal cyclicity of the mare. Though the investigators did not assess muscle mass, the anabolic steroid treatment did not have an effect on weight gain or height at the withers. The study concluded that due to the detrimental effects, anabolic steroids are not recommended for use in mares intended for reproductive use. Additional studies have noted that both mares and stallions exhibit altered sexual behavior due to anabolic steroid injections (Squires et al., 1982; 1983), however growth rates are not affected (Burke et al., 1981). Though RAC is not a hormone, the results of its administration, such as the depletion of adipocytes in extreme cases, could prove to have a negative effect on reproductive performance.

A Comparison to Clenbuterol (CBL)

Clenbuterol HCl, a β-agonist of the same class as RAC, has been shown to have similar repartitioning effects in horses as RAC (Kearns et al., 2001); however due to the presence of a halogenated aromatic ring, it has a longer half life than does RAC with a hydroxylated aromatic ring (Smith, 1998). Though clenbuterol is primarily administered as a therapeutic agent to resolve bronchospasm and chronic obstructive pulmonary disease (COPD) in race horses, an accelerated rate of supplementation can result in repartitioning effects. Unfortunately, like many drugs, its abuse can lead to increased or abnormal heart rates and other cardiac irregularities. These adverse effects could cause a horse to be more susceptible to stress. Due to clenbuterol's similarities as a β-agonist, the same concerns should be evaluated to determine the safety of RAC when administered in horse diets. Furthermore, data have revealed that RAC-supplemented pigs are potentially more susceptible to handling and transport stress due to hyperactivity, and higher heart rates during transport (Marchant-Forde et al., 2003). It has been well accepted by the swine community that special care should be taken when handling RAC-supplemented pigs to reduce the risk of "Downer Pigs."

A Reproductive Perspective

Although RAC is not currently recommended for use in breeding animals, there is little evidence identifying potential effects on reproduction. An unpublished report (Williams, 1989 as found in Ungemach, 2004) concerning crossbred swine fed ractopamine suggested that the reproductive performance of gilts would not be negatively affected after supplementation ceased. Gilts were fed a diet containing either 20 or

60mg/kg BW of RAC. Following withdrawal, gilts were bred, allowed to farrow and nurse. There was no effect determined on reproductive performance. However, due to inappropriate standards of protocol, this study was not deemed suitable to assess the safety of ractopamine. Though RAC is not expected to be directly involved hormonally like a steroid, the dramatic increase in muscle gain and decrease in adipose tissue, as seen in swine, could potentially be adverse to normal reproductive physiology in the mare.

In extreme cases, the depletion of adipocytes in the mare could cause an indirect route of interference with the normal reproductive cycle. In many farm animal species such as cattle and sheep, dietary energy restriction can lead to impaired reproductive function such as a delayed onset of puberty, as well as the induction and prolonging of anestrus (Rutter and Randel, 1894; Schillo 1992). This is achieved by certain effects on the pituitary's function to release gonadatropins. Nutrition and reproductive efficiency has been correlated in the horse (Henneke et al., 1983, 1984; Hines et al., 1987). However, in the mare, the relationship between nutritional status and reproductive hormone release is not entirely understood. Nevertheless, mares in a moderately fat condition withhold adequate energy reserves (body fat) to meet the high-energy demands of reproduction. Additionally, mares entering the breeding season in thin condition have lower pregnancy rates and a higher number of cycles per conception (Henneke et al., 1984). In more recent years, research has further determined that the continuous reproductive activity exhibited by some mares is associated with a higher concentration of leptin, body weight, and an estimated percent body fat (Ferreira-Dias et al., 2002). First discovered in the mouse, leptin is an "adipostatic" hormone that regulates multiple metabolic processes including fertility (Henson et al., 2003). Leptin secretion can be

down-regulated by multiple factors including fasting, decreased adiposity, β-agonists, and growth hormone (Ahren et al., 1997; Considine and Caro, 1997; Houseknecht et al., 2000). However, leptin concentrations in the horse can be greatly variable due to their large body mass. A species with a large body mass has a greater availability of oxidizable energy stores to counteract the negative response of the hypothalamic-pituitary-gonadal axis to feed reduction (McManus and Fitzgerald, 2000). As leptin has been deemed as the signal between body fat and the hypothalamus (Houseknecht et al., 1998), it is possible for gonadatropin secretion (LH and FSH) to be affected. For instance, dietary energy restriction in cattle and sheep suppresses the episodic release of LH (Schillo, 1992). As well, the absence of ovulation and the inability to form a functional corpus luteum has been correlated with low body condition in mares (Gentry et al., 2002). A study conducted by McManus and Fitzgerald (2000) demonstrated that circulating levels of leptin decreased in both young and aged mares when placed under a short term feed restriction; however, serum levels of LH and FSH were not affected during the same study.

Current Regulations

The use of clenbuterol and RAC is banned by the United States Equestrian Federation which is the governing body of all equine performance activities (U.S. Equestrian Federation, 2007), as well as the Association of Racing Commissioners, International as a ‘Class 2 Drug”, having a high potential for affecting the performance of the horse (ARCI, 2006). Despite current regulations on the use of RAC as a performance enhancing drug in equine entities, speculation exists that many owners and trainers insist

on its off-label use in horses. Detection of RAC and its metabolites is achieved through performing an enzyme-linked immunosorbent assay (ELISA) of horse urine (Lehner et al., 2004)

Thus far, only anecdotal evidence has suggested the efficacy of RAC supplementation to horse diets to induce weight loss. Published research does not provide an in depth investigation to changes in physical parameters in the horse, as compared to publications in both swine and cattle. Furthermore, little is known about the effects RAC could have on normal reproductive physiology within any species.

CHAPTER III

MATERIALS AND METHODS

Management of Horses

Thirteen fillies of varying ages (2, 3, and 5 years old), of stock horse type, were used in this study. All horses utilized were property of the Texas A&M University Department of Animal Science, and were maintained by the staff and facilities of the Texas A&M University Horse Center and N.W. "Dick" Freeman Arena. Maintenance and general care of all horses throughout this study conformed to the guidelines set forth by the Institutional Agricultural Animal Care and Use Committee (AUP# 2006-106).

Horses were housed in approximately 3.1m X 3.1m stalls, and received a moderate level of exercise 5 d per wk. Additional lighting was utilized to serve as an artificial photoperiod to ensure 16 hr per d of light exposure consistent with the seasonal reproductive period in the horse. All horses received a commercially available concentrate (Producer's Cooperative Association, Bryan, TX) containing 13% crude protein at 1% BW per d. Feedings for each horse were adjusted weekly according to the recorded weekly BW. The remainder of the diet consisted of average quality coastal bermudagrass hay, and water was provided ad libitum. All feedings were conducted at 0630 and 1730 daily throughout the 90-d period.

Horses observed in the treatment group received the same diet as explained previously, however, with the addition of RAC HCl at a rate of 0.6mg/kg BW per d, which was also adjusted according to weekly changes in BW. This oral supplement was top dressed to the morning feed for each horse in the treatment group.

Experimental Procedure

The thirteen fillies were randomly divided into 2 dietary groups: the control group (n=7) and the treatment group (n=6). All horses began the experimental period on September 10, 2006 (d 0) and ended the experimental period on December 10, 2006 (d 90). All horses remained in their respective dietary group and received the appropriate diet, according to individual BW, throughout the 90-d period.

Physical Measurements

Body weights for each individual horse were recorded beginning on d 0, and then weekly for the remainder of the study. These measurements were recorded prior to feeding at the same time each day to minimize effects of digestive fill. Body weight measurements were further used to make weekly adjustments to diets for all horses, as well as the supplemental RAC HCl.

Forearm and gaskin circumference were measured weekly, beginning on d 0, at the widest point of each muscle group using a simple measuring tape. The left forearm and right gaskin muscles were observed to prevent any confounding effects of side-specific exercise. Additionally, the area measured was also clipped to assure the same point was measured during each collection period.

Rump fat and rib fat measurements were collected weekly, beginning on d 0, via ultrasonic scanning equipment with a 5MHz transducer (Medison SonoVet 600®, Universal, Bedford Hills, NY). Rump fat measurements for all horses were taken from an area central to the length of the hip and 5.00 cm from the midline. Rib fat measurements were collected from an area over the ribs, precisely between the point of the withers and

point of the hip, and 45.00cm lateral from the midline. Body fat percentage was calculated from the appropriate rump fat values obtained throughout the study.

Reproductive Measurements

Follicular growth and ovulation schedules were tracked via rectal palpation and ultrasound twice per wk, or every 3 to 4 d, beginning prior to d 0. The largest follicle on each ovary was measured and recorded via ultrasonic scanning equipment with a 5MHz transducer (Medison SonoVet 600®, Universal, Bedford Hills, NY). Additionally, the presence of a corpus luteum on the ovary was also recorded as an indicator of ovulation.

Serum Sampling

Blood was collected from each horse using jugular venipuncture beginning on d 0, and continuing on an every other day basis. Blood samples were collected at 0600 before feeding to minimize fluctuations in circulating hormone concentrations. Upon collection, blood samples were transported on ice in preparation for centrifugation. All samples were centrifuged within 2 h of collection in a refrigerated centrifuge. Centrifuge temperature was 5°C and samples were spun at 2500rpm for 30 min. Upon completion of centrifugation, serum was separated and stored in micro-centrifuge tubes at -20°C for subsequent hormone analysis.

Radioimmunoassay (RIA) Procedures

Serum concentrations of leptin and luteinizing hormone (LH) were analyzed via RIA for horse samples. All samples from each horse were analyzed in a single assay for

each hormone. All unknown assay samples were run in duplicate while standards and reagents were run in triplicate.

Serum leptin concentrations were measured on a weekly basis using a multi-species leptin RIA kit (Linco Research, Inc., St. Charles, MO.). A serum sample volume of 100µl was used with a sensitivity of 1.0 ng/ml. Leptin assays were analyzed using a Packard Cobra II® gamma counter. Location of assay was conducted at Texas A&M University – Kleberg Center, College Station, Texas.

Serum LH concentrations were measured on an every other day basis using e-LH RIA procedures (Williams et al., 2007). A serum sample volume of 200µl was used with a sensitivity of 0.10ng/ml. Luteinizing hormone assays were analyzed using a Perkin Elmer gamma counter. Assays were conducted in conjunction with Dr. Gary L. Williams and staff at the Animal Reproduction Laboratory, Texas Agrilife Research Station, Beeville, Texas.

Statistical Analyses

Statistical analysis was conducted on all physical parameters, including leptin, by analysis of variance (ANOVA) using STATA statistical software (Stata Corp., College Station, TX). Physical parameters were analyzed as the mean change from d 0 to establish a baseline. Raw data was initially analyzed for leptin; however, due to numeric variability between horses and groups at d 0, normalized values are also represented.

Data collected for length of estrous cycle, pre-ovulatory follicle size, and luteinizing hormone (LH) concentrations were analyzed using the mixed linear model (PROC MIXED) of SAS (2007). Least squares means and standard errors were obtained

according to treatment, cycle and treatment by cycle for length of estrous cycle and pre-ovulatory follicle size; where as treatment, day and treatment by day was utilized for LH. Main treatment effects were analyzed using horse within treatment as the error term.

Statistical significance was declared at probabilities <0.05 for all data, with probability values between 0.05 and 0.20 being considered to be trends towards significance.

CHAPTER IV

RESULTS

Physical Measurements

Body weight was measured on a weekly schedule not only to provide accurate RAC supplementation, but also to monitor the weekly response to RAC and to determine the overall change in body weight during the supplemental period. Statistical analysis of the data (Table 1) indicated an effect of RAC supplementation as treated horses had a greater increase in BW (P<.001) compared to the controls. Since both groups began at differing body weights, only the changes in means from d 0 to d 90 are represented in order to establish a baseline reference.

Table 1. Mean (± SE) body weight measured in mares fed a control or RAC-supplemented diet.

	Body Weight (kg)		
	Initial	Final	Change
Control	444.29 ± 8.7	461.95 ± 7.3	17.66[a] ± 3.8
Treatment	423.33 ± 20.3	448.18 ± 17.0	24.85[b] ± 6.5

[a,b] Means within columns with different superscripts differ (P<.001)

Muscle growth was monitored for both groups by measuring the circumference around the widest point of both the forearm and gaskin muscles. The point of measurement was shaved to ensure exact location of subsequent recordings, and measurement was performed by the same individual to decrease the likelihood of error in measuring. Though no change was observed in forearm circumference (Table 2) for either group, data indicated that treatment horses increased gaskin circumference by a

mean of 0.67 ± 0.2 cm (P<.001) compared to 0.00 ± 0.0 cm for the horses on the control diet (Table 3).

Table 2. Mean (± SE) forearm circumference measured in mares fed a control or RAC-supplemented diet.

	Forearm Circumference (cm)		
	Initial	Final	Change
Control	53.33 ± 0.5	53.51 ± 0.6	0.19 ± 0.2
Treatment	52.12 ± 1.0	52.58 ± 1.2	0.47 ± 0.5

Table 3. Mean (± SE) gaskin circumference measured in mares fed a control or RAC-supplemented diet.

	Gaskin Circumference (cm)		
	Initial	Final	Change
Control	45.97 ± 0.6	45.97 ± 0.5	0.00^{a} ± 0.0
Treatment	44.53 ± 0.9	45.20 ± 0.9	0.67^{b} ± 0.2

a,b Means within columns with different superscripts differ (P<.001)

Although there was no difference in the change in rib fat (Table 4), both groups showed an increase in rump fat (Table 5) with the treated horses gaining less rump fat (P<.05). A similar effect was revealed in body fat percentage (P<.01) with the treated horses gaining less when compared to the controls (Table 6). This can be expected as rump fat values were utilized in calculation of percent body fat values according to the equation: Y= 8.64 + 4.70 X (Westervelt et al., 1976). As both groups indicated similar loss of rib fat, this parameter could be attributed to the exercise requirements under saddle.

Table 4. Mean (± SE) rib fat measured in mares fed a control or RAC-supplemented diet.

	Rib Fat (mm)		
	Initial	Final	Change
Control	4.14 ± 0.6	3.14 ± 0.1	-1.00 ± 0.6
Treatment	3.83 ± 0.3	2.83 ± 0.2	-1.00 ± 0.4

Table 5. Mean (± SE) rump fat measured in mares fed a control or RAC-supplemented diet.

	Rump Fat (mm)		
	Initial	Final	Change
Control	5.43 ± 0.9	9.29 ± 1.1	3.86[a] ± 0.7
Treatment	4.83 ± 0.4	7.33 ± 0.8	2.50[b] ± 0.6

[a,b] Means within columns with different superscripts differ (P<.05)

Table 6. Mean (± SE) percent body fat measured in mares fed a control or RAC-supplemented diet.

	Body Fat (%)		
	Initial	Final	Change
Control	11.20 ± 0.4	12.99 ± 0.5	0.93[a] ± 0.1
Treatment	10.92 ± 0.2	12.07 ± 0.4	0.73[b] ± 0.1

[a,b] Means within columns with different superscripts differ (P<.01)

Reproductive Measurements

Mean estrous cycle length was counted in days from one ovulation to the next throughout the duration of the study. Ovulation was monitored twice per week beginning at the first recorded corpus luteum (CL) to the presence of a subsequent CL. Mean pre-

ovulatory follicle size was measured and was the mean (mm) of the largest follicle measured before ovulation for each group. Initial values were taken beginning on d 0 and final values ended at d 90. Upon analysis of length of estrous cycle, there were no treatment or treatment by cycle effects; however, treatment horses did experience a shorter estrous cycle at the conclusion of the study as compared to initial cycle length within that group (Table 7). On average, there was no difference associated with mean length of cycle between groups throughout the study. There were no observed effects for pre-ovulatory follicle size, although numerically, treatment horses on average sustained a larger pre-ovulatory follicle size for the entire study (Table 8).

Table 7. Mean (± SE) length of estrous cycle in mares fed a control or RAC supplemented diet.

	Length of Estrous Cycle (d)		
	First Cycle	Last Cycle	Mean Cycle Length
Control	24.29 ± 5.9	21.70 ± 1.9	23.11 ± 1.6
Treatment	33.43 ± 5.9[a]	15.16 ± 2.1[b]	23.15 ± 1.7

[a,b] Means within rows with different superscripts differ (P<.05)

Table 8. Mean (± SE) pre-ovulatory follicle size in mares fed a control or RAC supplemented diet.

	Pre-Ovulatory Follicle Size (mm)		
	First Cycle	Last Cycle	Mean Size
Control	35.57 ± 1.7	38.67 ± 2.3	35.99 ± 1.1
Treatment	37.00 ± 1.7	34.00 ± 2.8	36.34 ± 1.2

Hormone Analyses: Serum Leptin

Serum Leptin concentrations were measured on a weekly schedule beginning on d 0 to further support the results concluded from body fat measurements and to determine if RAC supplementation had an effect on circulating hormone concentrations. Though blood samples were taken every other day, a weekly sample (same day for all horses) was utilized to determine the weekly fluctuation of the leptin concentration profile. Statistical analysis of raw data indicated an effect (P<.001) of RAC on circulating levels of leptin with the treatment group having a lower total concentration at the end of the 90-d period (Table 9).

Table 9. Mean (± SE) serum leptin concentrations (ng/ml) in mares fed a control or RAC-supplemented diet.

Week	Control	Treatment
0	2.7 ± 0.7	1.6 ± 0.2
1	3.2 ± 0.8	1.9 ± 0.4
2	2.8 ± 0.7	1.9 ± 0.3
3	2.7 ± 0.7	1.7 ± 0.3
4	2.8 ± 0.7	2.0 ± 0.4
5	2.9 ± 0.8	1.9 ± 0.3
6	3.0 ± 0.8	2.1 ± 0.3
7	3.0 ± 0.8	1.8 ± 0.4
8	3.0 ± 0.8	2.0 ± 0.4
9	3.1 ± 0.9	2.1 ± 0.4
10	3.0 ± 0.9	2.0 ± 0.4
11	3.0 ± 0.9	2.2 ± 0.5
12	3.2 ± 0.9	2.3 ± 0.4
13	2.9 ± 0.9	2.1 ± 0.4
Overall Total	2.9^{a} ± 0.2	2.0^{b} ± 0.1

[a,b] Means between columns with different superscripts differ (P<.001).

However, this lower concentration was already present at the beginning of the study, but not significantly different between groups. Due to this slight variability

between groups at d 0, normalized values are represented to more accurately define the change in parameter (Figure 1). There were no significant differences in circulating leptin concentrations observed between groups using normalized values (Figure 1.) Nevertheless, this figure does represent the variability of circulating leptin concentrations noted in horses, and could possibly depict a trend that RAC may cause the leptin profile to become more erratic.

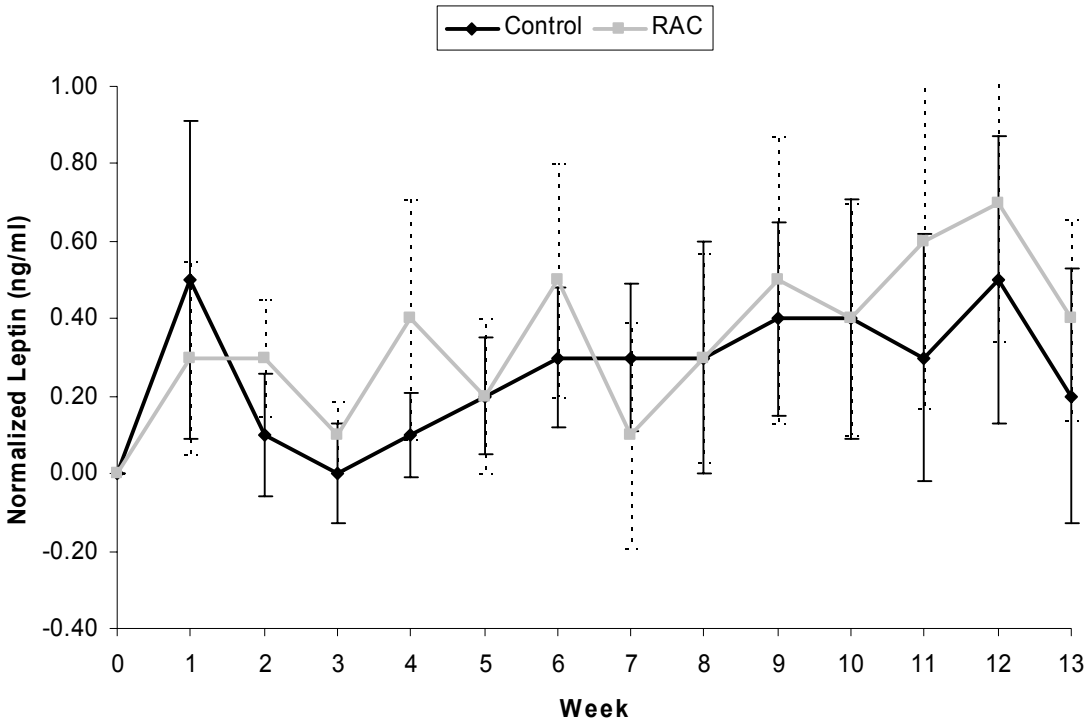

Figure 1. Normalized analysis of change in serum leptin concentrations measured in mares fed a control or RAC-supplemented diet.

Hormone Analyses: Serum Luteinizing Hormone (LH)

Serum Luteinizing Hormone (LH) concentrations were measured every other day beginning on d 0 and ending on d 90 to verify ovulation and determine changes during the season and the supplemental period. Analysis of means between each group for each day were plotted (Figure 2.), and a strong trend (P=0.0527) was noted for RAC-supplemented horses having a lower mean concentration of LH (0.88 ± .22) throughout the 90-d study as compared to the controls (1.53 ± .20).

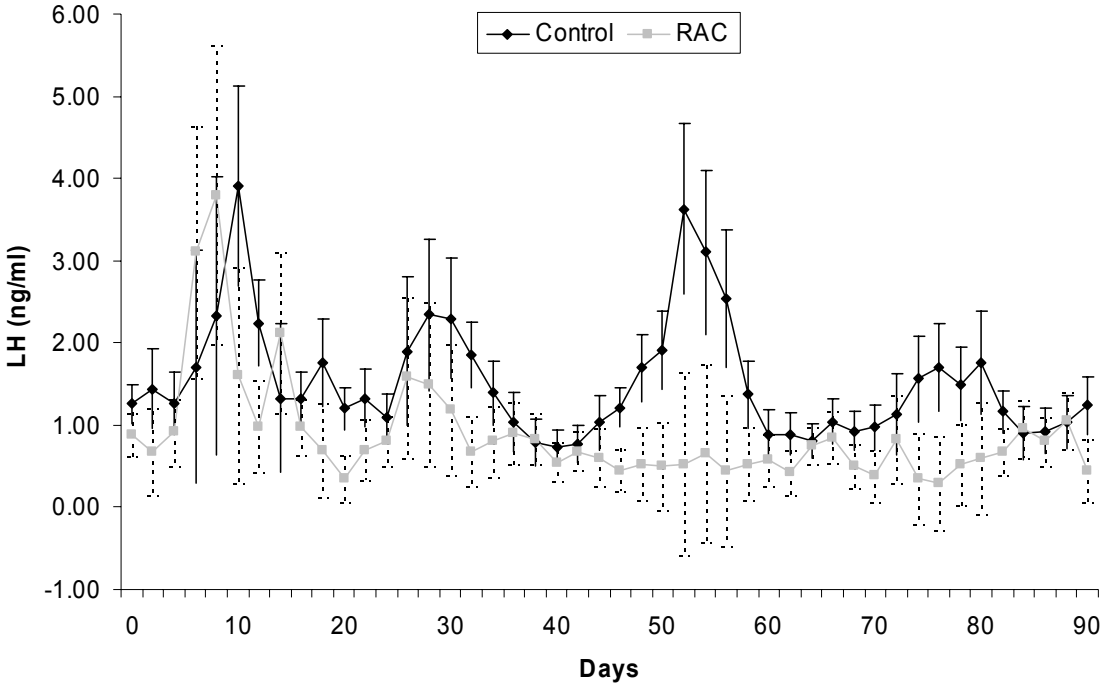

Figure 2. Mean luteinizing hormone (ng/ml) concentrations measured in mares fed a control or RAC-supplemented diet.

Means were also analyzed for day (P=0.2944) and treatment by day (P=0.1591) effects, suggesting a possible seasonal effect throughout the study. Due to the season in which the study was conducted (September-December), season could be a confounding variable. All horses were placed under an artificial photoperiod to assist in minimizing this effect, however not all horses responded similarly to the lighting regimen. In any case, the control horses appeared to numerically maintain a higher concentration of LH, depicted as the spike around d 52, while the RAC-supplemented horses remained suppressed (Figure 2) after the initial LH peak.

Area Under the Curve (AUC) was calculated for individual horses and analyzed for treatment effects by analysis of variance. Only a trend (P=0.1631) was noted for RAC-supplemented horses having a smaller AUC (80.10 ± 29.72) as compared to the controls (140.60 ± 27.50).

CHAPTER V

DISCUSSION

Previously, only anecdotal evidence has suggested the possible efficacy of RAC usage in horse diets. Published research does not provide an in depth investigation in to changes in physical parameters in the horse, as compared to publications in both swine and cattle. Although RAC is not recommended for use in breeding animals, there have been no reports identifying potential effects on reproduction. However, the dramatic increase in muscle gain and decrease in adipose tissue, as seen in swine, could potentially be adverse to normal reproductive physiology.

The results of this study show similar trends to data obtained from swine fed RAC in the form of Paylean®. Prior studies in swine commonly note that consumption of RAC at differing levels resulted in increases in BW and percent yield and lean, which appears to agree with this study (Kelly et al., 2003; See et al., 2004). The current study contradicts the decrease in BW as seen in obese pony mares fed RAC. However, those ponies were restricted to 75% of ad libitum intake, whereas the current study did not feed for the same negative energy balance that would initiate weight loss.

Leptin concentrations for obese pony mares (Buff et al., 2006) changed significantly over time; however there was no difference observed between groups. The current study indicates a change in mean overall serum leptin concentrations. Although, there was slight variability at the beginning of the study, and normalized data did not indicate any change between groups. Even so, it appears that RAC supplementation may have caused leptin to become more erratic over time. Additionally, data collected for LH concentrations indicated a strong trend for RAC-supplemented horses to have a lower

mean concentration throughout the 90-d period. As well, there were trends for day and treatment by day effects, suggesting a possible seasonal effect. Horses were placed under an artificial photoperiod, though not all horses responded as expected. In any case, it would appear that RAC may have caused a suppression of LH for the treatment horses as the control group numerically maintained higher levels of LH throughout the study.

Administration of RAC in horse diets resulted in significant increases in BW and gaskin circumference, without affecting follicular growth and ovulation. However, restrictions are being placed on the use of RAC as a possible performance enhancing drug by the Association of Racing Commissioners, International among others (ARCI, 2006; U.S. Equestrian Federation, 2007). Clenbuterol HCl, another β-agonist that is used in horses as a therapeutic drug for bronchospasm and chronic obstructive pulmonary disease (COPD), has also been shown to have legitimate repartitioningeffects similar to RAC (Kearns et al., 2001). Although this drug has therapeutic intentions, the abuse of clenbuterol can lead to increased or abnormal heart rates and other cardiac irregularities, thus horses are more susceptible to stressors. Additionally, RAC supplemented pigs must be handled by less stressful methods for the same reasons.

Coupled with the increased protein synthesis in muscle tissue, lysine deficiencies should also be of concern. Eli Lilly and Company (Elanco), which markets Paylean® and Optaflexx®, recommends feeding at least a 16% CP ration to pigs in order to prevent a lysine deficiency and support the accelerated protein synthesis to optimize the performance of RAC fed swine. However, lysine requirements and the lysine:energy value are thought to be greater in RAC-supplemented pigs and may be higher than what is currently being used in the industry (Apple et al., 2004). It appears that a diet

consisting of at least 1.0% dietary lysine will be suitable to optimize growth rate and lean yield in swine (Webster et al., 2002).

As the industry becomes more competitive and more is expected from younger horses, meeting their nutritional requirements becomes a challenge. If such a product is used in younger, halter horses for example, an average diet could be lacking sufficient levels of lysine to support growth and development. Thus, further research should be conducted to reveal the safety of RAC supplemented diets in horses, and to evaluate any deficiencies or toxicities that may arise.

CHAPTER VI

SUMMARY

The present study monitored the effects of a RAC-supplemented diet on both physical and reproductive parameters in growing mares. Treatment horses were fed both the diet and supplement according to body weight in order to minimize non-RAC sources of variation. Physical measurements (BW, forearm circumference, and gaskin circumference) were recorded to monitor overall growth and muscle accretion. Additionally, fat thickness was measured (rib fat, rump fat, and percent body fat) to gauge the repartitioning effects of lean muscle growth. To help satisfy the concerns on reproduction, fillies were palpated to track follicular growth, ovulation, and corpus luteum maintenance. To further support the above information, serum samples were taken to monitor weekly changes in circulating leptin, and daily changes in circulating LH. Data collected was then analyzed to ensure accurate understanding of the parameters of concern.

The results of this study indicate a lean muscle gain and decrease in body fat percentage in horses fed RAC, a response similar to that seen in swine and cattle. Body weight increased as a result of muscle gain and overall growth versus fat deposition. However, if RAC is to be used in horse diets, safe levels of supplementation to avoid possible protein deficiencies and cardiovascular abnormalities should be considered.

In conclusion, the results obtained and presented in this study have provided the industry a closer look into the effects of RAC in horse diets as a repartitioning agent. Ractopamine does, in fact, initiate lean muscle growth even in smaller doses as compared to the typical doses supplemented to swine. Though no effects were noted for length of

estrous cycle or pre-ovulatory follicle size, RAC supplementation did appear to have an effect on mean serum leptin concentrations (no effect for normalized data), and multiple trends were noted for LH concentrations for horses supplemented with RAC. The results presented for hormone analyses are somewhat unclear due to possible seasonal effects. As this supplemental period was short-term, and again RAC was supplemented at relatively low levels, the effects of long-term and accelerated dosage levels on reproduction should still be questioned. Nevertheless, the analysis and results presented within this study illustrate RAC's capability of altering growth and possibly performance in horses to the governing bodies of the equine industry.

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APPENDIX

Appendix Table 1A. Description of research horses.

Horse	Age	Sex	Breed	Treatment
Dee	2	Filly	Quarter Horse	RAC
Missy	5	Filly	Quarter Horse	RAC
Mark Kay	3	Filly	Quarter Horse	RAC
Blue	5	Filly	Quarter Horse	RAC
Scoot	2	Filly	Quarter Horse	RAC
Prissy	2	Filly	Quarter Horse	RAC
Total (RAC)				n=6
Sun	2	Filly	Quarter Horse	Control
Nina	3	Filly	Quarter Horse	Control
Marla	3	Filly	Quarter Horse	Control
Faces	2	Filly	Quarter Horse	Control
Loreal	2	Filly	Quarter Horse	Control
Slimer	2	Filly	Quarter Horse	Control
Lena	3	Filly	Quarter Horse	Control
Total (Control)				n=7

Appendix Table 2A. ANOVA table for change in body weight (BW).

Source	DF	Partial SS	Mean Square	F Value	Pr>F
Model	27	13819.2851	511.825374	5.86	0.0000
Group	1	1386.55166	1386.55166	15.88	0.0001
Week	13	11617.915	893.685769	10.23	0.0000
Group*Week	13	1238.53908	95.2722373	1.09	0.3707
Residual	154	13448.0086	87.3247314		
Total	181	27267.2937	150.648032		

Appendix Table 3A. ANOVA table for change in forearm circumference.

Source	DF	Partial SS	Mean Square	F Value	Pr>F
Model	27	5.17111709	0.191522855	0.27	0.9999
Group	1	0.10477369	0.10477369	0.15	0.7020
Week	13	4.00194787	0.307842144	0.43	0.9561
Group*Week	13	1.31029949	0.100792268	0.14	0.9998
Residual	154	109.792619	0.712939083		
Total	181	114.963736	0.635158762		

Appendix Table 4A. ANOVA table for change in gaskin circumference.

Source	DF	Partial SS	Mean Square	F Value	Pr>F
Model	27	10.4113736	0.385606429	3.73	0.0000
Group	1	6.0584825	6.0584825	58.61	0.0000
Week	13	3.18734165	0.245180127	2.37	0.0064
Group*Week	13	1.38909992	0.10685384	1.03	0.4221
Residual	154	15.9185715	0.103367348		
Total	181	26.3299451	0.14546931		

Appendix Table 5A. ANOVA table for change in rib fat thickness.

Source	DF	Partial SS	Mean Square	F Value	Pr>F
Model	27	44.1611722	1.63559897	1.17	0.2700
Group	1	2.0115123	2.0115123	1.44	0.2319
Week	13	33.8859236	2.60660951	1.87	0.0380
Group*Week	13	9.09471481	0.699593447	0.50	0.9214
Residual	154	215.047619	1.39641311		
Total	181	259.208791	1.43209277		

Appendix Table 6A. ANOVA table for change in rump fat thickness.

Source	DF	Partial SS	Mean Square	F Value	Pr>F
Model	27	282.576923	10.465812	4.64	0.0000
Group	1	14.1602564	14.1602564	6.28	0.0132
Week	13	247.999084	19.0768526	8.46	0.0000
Group*Week	13	13.9990842	1.07685263	0.48	0.9344
Residual	154	347.071429	2.25371058		
Total	181	629.648352	3.47872017		

Appendix Table 7A. ANOVA table for change in body fat percentage.

Source	DF	Partial SS	Mean Square	F Value	Pr>F
Model	27	3.2873076	0.121752133	1.68	0.0272
Group	1	0.603141005	0.603141005	8.31	0.0045
Week	13	2.47999075	0.19076852	2.63	0.0024
Group*Week	13	0.13999086	0.010768528	0.15	0.9998
Residual	154	11.1707139	0.072537103		
Total	181	14.4580215	0.079878572		

Appendix Table 8A. ANOVA table for length of estrous cycle.

Source	DF	Partial SS[a]	Mean Square[a]	F Value	Pr>F
Group	1			0.00	0.9873
Cycle	3			3.97	0.0198
Group*Cycle	3			1.46	0.2504

[a]Columns with superscript: values not calculated under Mixed Procedure for SAS.

Appendix Table 9A. ANOVA table for pre-ovulatory follicle size.

Source	DF	Partial SS[a]	Mean Square[a]	F Value	Pr>F
Group	1			0.04	0.8413
Cycle	3			0.18	0.9063
Group*Cycle	3			0.99	0.4146

[a]Columns with superscript: values not calculated under Mixed Procedure for SAS.

Appendix Table 10A. ANOVA table for change in serum leptin concentrations.

Source	DF	Partail SS	Mean Square	F Value	Pr>F
Model	27	47.8002209	1.77037855	0.60	0.9379
Group	1	42.6622459	42.6622459	14.54	0.0002
Week	13	4.19241046	0.322493112	0.11	1.0000
Group*Week	13	0.939813812	0.07229337	0.02	1.0000
Residual	154	451.818414	2.9338858		
Total	181	499.618635	2.76032395		

Appendix Table 11A. ANOVA table for normalized change in serum leptin concentrations.

Source	DF	Partial SS	Mean Square	F Value	Pr>F
Model	27	5.37812341	0.199189756	0.44	0.9934
Group	1	0.240148356	0.240148356	0.52	0.4700
Week	13	4.19241044	0.322493111	0.70	0.7569
Group*Week	13	0.939813882	0.072293376	0.16	0.9997
Residual	154	70.4943297	0.457755387		
Total	181	75.8724531	0.419184824		

Appendix Table 12A. ANOVA table for mean serum LH concentrations.

Source	DF	Sum of Squares[a]	Mean Square[a]	F Value	Pr>F
Group	1			4.71	0.0527
Week	45			1.11	0.2944
Group*Week	45			1.22	0.1591

[a]Columns with superscript: values not calculated under Mixed Procedure for SAS.

Appendix Table 13A. ANOVA table for serum LH concentrations (AUC).

Source	DF	Sum of Squares	Mean Square	F Value	Pr>F
Model	1	11825.42308	11825.42308	2.23	0.1631
Error	11	58226.4400	5293.31237		
Corrected Total	12	70051.863.08			

Appendix Table 14A. Physical data collected on project horses.

Horse	Group	Week	BW (kg)	Forearm (cm)	Gaskin (cm)	Rib Fat (mm)	Rump Fat (mm)	Rump Fat (cm)	Body Fat (%)
Dee	RAC	0	434.09	51.0	44.5	4	4	0.4	10.5
Dee	RAC	1	436.36	51.0	44.0	4	4	0.4	10.5
Dee	RAC	2	433.18	51.2	44.1	4	5	0.5	11.0
Dee	RAC	3	444.09	51.2	44.1	4	4	0.4	10.5
Dee	RAC	4	448.18	51.2	44.1	3	4	0.4	10.5
Dee	RAC	5	449.55	51.2	44.1	3	4	0.4	10.5
Dee	RAC	6	450.91	51.2	44.1	3	5	0.5	11.0
Dee	RAC	7	458.64	51.3	44.1	2	6	0.6	11.5
Dee	RAC	8	460.00	51.5	44.1	2	7	0.7	11.9
Dee	RAC	9	462.27	51.7	44.3	2	7	0.7	11.9
Dee	RAC	10	466.82	51.9	44.3	2	7	0.7	11.9
Dee	RAC	11	480.45	52.0	44.5	3	8	0.8	12.4
Dee	RAC	12	470.91	52.0	44.5	3	7	0.7	11.9
Dee	RAC	13	469.55	52.0	44.6	2	7	0.7	11.9
Missy	RAC	0	436.36	53.4	44.8	3	5	0.5	11.0
Missy	RAC	1	454.55	53.4	44.9	2	5	0.5	11.0
Missy	RAC	2	432.73	53.6	45.3	2	5	0.5	11.0
Missy	RAC	3	453.64	53.7	45.4	2	5	0.5	11.0
Missy	RAC	4	457.27	53.9	45.6	2	5	0.5	11.0
Missy	RAC	5	455.45	53.9	45.6	2	6	0.6	11.5
Missy	RAC	6	460.00	54.1	45.6	2	6	0.6	11.5
Missy	RAC	7	456.82	54.2	45.7	2	6	0.6	11.5
Missy	RAC	8	460.45	54.2	45.7	2	7	0.7	11.9
Missy	RAC	9	461.36	54.2	45.7	3	7	0.7	11.9
Missy	RAC	10	460.00	54.3	45.7	3	7	0.7	11.9
Missy	RAC	11	476.36	54.4	45.8	2	9	0.9	12.9
Missy	RAC	12	465.91	54.4	45.9	3	7	0.7	11.9
Missy	RAC	13	471.36	54.6	46.1	3	7	0.7	11.9

Appendix Table 14A (Continued). Physical data collected on project horses.

Horse	Group	Week	BW (kg)	Forearm (cm)	Gaskin (cm)	Rib Fat (mm)	Rump Fat (mm)	Rump Fat (cm)	Body Fat (%)
Mary Kay	RAC	0	479.09	55.1	48.4	4	6	0.4	10.5
Mary Kay	RAC	1	484.09	55.1	48.4	4	9	0.4	10.5
Mary Kay	RAC	2	479.55	55.2	48.4	5	11	0.5	11.0
Mary Kay	RAC	3	490.00	55.2	48.4	4	9	0.4	10.5
Mary Kay	RAC	4	463.64	55.4	48.4	3	8	0.3	10.1
Mary Kay	RAC	5	487.27	55.4	48.4	2	8	0.2	9.6
Mary Kay	RAC	6	486.82	55.6	48.4	3	7	0.3	10.1
Mary Kay	RAC	7	495.45	55.7	48.5	2	8	0.2	9.6
Mary Kay	RAC	8	501.36	55.9	48.5	3	10	0.3	10.1
Mary Kay	RAC	9	507.73	56.0	48.5	3	11	0.3	10.1
Mary Kay	RAC	10	495.45	56.0	48.5	3	10	0.3	10.1
Mary Kay	RAC	11	507.25	56.1	48.7	3	12	0.3	10.1
Mary Kay	RAC	12	499.55	56.1	48.7	3	9	0.3	10.1
Mary Kay	RAC	13	492.73	56.1	48.7	3	10	0.3	10.1
Blue	RAC	0	464.55	54.0	44.7	4	4	0.4	10.5
Blue	RAC	1	458.09	54.0	44.9	3	4	0.3	10.1
Blue	RAC	2	447.73	52.0	44.9	4	6	0.4	10.5
Blue	RAC	3	455.45	52.2	45.1	4	6	0.4	10.5
Blue	RAC	4	477.27	52.2	45.1	2	4	0.2	9.6
Blue	RAC	5	456.36	52.2	45.1	3	4	0.3	10.1
Blue	RAC	6	453.18	52.3	45.0	2	4	0.2	9.6
Blue	RAC	7	455.00	52.3	45.1	2	4	0.2	9.6
Blue	RAC	8	460.00	52.3	45.2	2	5	0.2	9.6
Blue	RAC	9	460.91	52.3	45.2	2	5	0.2	9.6
Blue	RAC	10	454.54	52.3	45.3	3	5	0.3	10.1
Blue	RAC	11	476.82	52.3	45.3	3	5	0.3	10.1
Blue	RAC	12	479.55	52.4	45.3	3	4	0.3	10.1
Blue	RAC	13	462.27	52.5	45.4	3	4	0.3	10.1

Appendix Table 14A (Continued). Physical data collected on project horses.

Horse	Group	Week	BW (kg)	Forearm (cm)	Gaskin (cm)	Rib Fat (mm)	Rump Fat (mm)	Rump Fat (cm)	Body Fat (%)
Scoot	RAC	0	359.55	48.5	41.5	3	4	0.4	10.5
Scoot	RAC	1	364.09	47.6	41.7	4	4	0.4	10.5
Scoot	RAC	2	366.82	47.6	41.8	3	4	0.4	10.5
Scoot	RAC	3	375.00	47.5	41.8	3	3	0.3	10.1
Scoot	RAC	4	375.45	47.5	41.8	2	4	0.4	10.5
Scoot	RAC	5	374.55	47.5	41.8	2	5	0.5	11.0
Scoot	RAC	6	372.73	47.5	41.9	2	5	0.5	11.0
Scoot	RAC	7	380.45	47.5	42.0	2	5	0.5	11.0
Scoot	RAC	8	383.18	47.5	42.2	2	5	0.5	11.0
Scoot	RAC	9	386.36	47.5	42.2	2	5	0.5	11.0
Scoot	RAC	10	385.91	47.5	42.2	2	6	0.6	11.5
Scoot	RAC	11	394.55	47.5	42.2	2	8	0.8	12.4
Scoot	RAC	12	390.00	47.6	42.3	2	8	0.8	12.4
Scoot	RAC	13	388.64	47.7	42.4	3	7	0.7	11.9
Prissy	RAC	0	366.36	50.7	43.3	5	6	0.6	11.5
Prissy	RAC	1	366.36	51.4	43.3	3	5	0.5	11.0
Prissy	RAC	2	369.55	51.4	43.2	4	7	0.7	11.9
Prissy	RAC	3	379.55	51.4	43.2	4	7	0.7	11.9
Prissy	RAC	4	384.09	51.4	43.2	4	8	0.8	12.4
Prissy	RAC	5	380.91	51.5	43.4	2	8	0.8	12.4
Prissy	RAC	6	384.55	51.7	43.6	2	9	0.9	12.9
Prissy	RAC	7	390.91	52.0	43.7	2	9	0.9	12.9
Prissy	RAC	8	396.36	52.0	43.7	2	9	0.9	12.9
Prissy	RAC	9	400.00	52.0	43.8	2	9	0.9	12.9
Prissy	RAC	10	403.18	52.0	43.9	2	9	0.9	12.9
Prissy	RAC	11	413.64	52.0	44.0	3	11	1.1	13.8
Prissy	RAC	12	402.27	52.4	44.0	3	8	0.8	12.4
Prissy	RAC	13	404.55	52.6	44.0	3	9	0.9	12.9

Appendix Table 14A (Continued). Physical data collected on project horses.

Horse	Group	Week	BW (kg)	Forearm (cm)	Gaskin (cm)	Rib Fat (mm)	Rump Fat (mm)	Rump Fat (cm)	Body Fat (%)
Sun	Control	0	440.91	52.0	45.5	1	7	0.7	11.9
Sun	Control	1	441.36	52.9	45.5	3	7	0.7	11.9
Sun	Control	2	430.45	52.9	45.5	3	6	0.6	11.5
Sun	Control	3	444.09	52.9	45.7	2	6	0.6	11.5
Sun	Control	4	446.82	52.9	45.7	2	6	0.6	11.5
Sun	Control	5	445.00	52.9	45.7	3	7	0.7	11.9
Sun	Control	6	445.00	52.5	45.7	2	7	0.7	11.9
Sun	Control	7	440.91	52.5	45.7	2	7	0.7	11.9
Sun	Control	8	453.18	52.5	45.7	2	8	0.8	12.4
Sun	Control	9	452.27	52.5	45.7	2	7	0.7	11.9
Sun	Control	10	452.27	52.5	45.5	2	7	0.7	11.9
Sun	Control	11	452.27	52.5	45.5	3	9	0.9	12.9
Sun	Control	12	454.09	52.5	45.5	3	9	0.9	12.9
Sun	Control	13	453.18	52.5	45.5	3	9	0.9	12.9
Nina	Control	0	463.18	53.6	45.1	4	10	1	13.3
Nina	Control	1	467.27	53.6	45.1	4	12	1.2	14.3
Nina	Control	2	466.36	54.0	45.1	4	13	1.3	14.8
Nina	Control	3	475.45	54.3	45.2	4	13	1.3	14.8
Nina	Control	4	466.82	54.3	45.3	4	13	1.3	14.8
Nina	Control	5	478.18	54.3	45.3	4	13	1.3	14.8
Nina	Control	6	475.91	54.4	45.3	4	13	1.3	14.8
Nina	Control	7	482.27	54.5	45.3	4	14	1.4	15.2
Nina	Control	8	487.27	54.5	45.3	4	15	1.5	15.7
Nina	Control	9	488.64	54.5	45.3	4	15	1.5	15.7
Nina	Control	10	486.36	54.5	45.3	3	15	1.5	15.7
Nina	Control	11	488.64	54.5	45.3	3	15	1.5	15.7
Nina	Control	12	494.09	54.5	45.3	3	16	1.6	16.2
Nina	Control	13	491.82	54.5	45.3	3	15	1.5	15.7

Appendix Table 14A (Continued). Physical data collected on project horses.

Horse	Group	Week	BW (kg)	Forearm (cm)	Gaskin (cm)	Rib Fat (mm)	Rump Fat (mm)	Rump Fat (cm)	Body Fat (%)
Marla	Control	0	485.00	54.6	49.1	5	6	0.6	11.5
Marla	Control	1	486.36	54.6	48.7	4	8	0.8	12.4
Marla	Control	2	477.27	54.6	48.8	4	8	0.8	12.4
Marla	Control	3	493.64	54.8	48.9	4	7	0.7	11.9
Marla	Control	4	495.45	54.9	49.1	4	8	0.8	12.4
Marla	Control	5	495.45	54.9	49.1	4	8	0.8	12.4
Marla	Control	6	488.64	54.9	49.1	4	7	0.7	11.9
Marla	Control	7	495.45	54.9	49.1	4	7	0.7	11.9
Marla	Control	8	495.45	55.0	49.0	4	8	0.8	12.4
Marla	Control	9	498.64	55.0	49.0	4	8	0.8	12.4
Marla	Control	10	490.91	55.0	49.0	3	8	0.8	12.4
Marla	Control	11	491.82	55.0	49.0	3	8	0.8	12.4
Marla	Control	12	489.55	55.0	49.0	3	8	0.8	12.4
Marla	Control	13	487.27	55.0	49.0	4	7	0.7	11.9
Faces	Control	0	415.45	53.9	45.1	4	3	0.3	10.1
Faces	Control	1	418.64	53.9	44.9	4	5	0.5	11.0
Faces	Control	2	406.82	53.9	45.0	3	4	0.4	10.5
Faces	Control	3	415.91	54.1	45.3	4	3	0.3	10.1
Faces	Control	4	421.82	54.1	45.3	5	4	0.4	10.5
Faces	Control	5	419.09	54.1	45.3	4	3	0.3	10.1
Faces	Control	6	422.73	54.2	45.3	4	3	0.3	10.1
Faces	Control	7	426.36	54.3	45.4	4	5	0.5	11.0
Faces	Control	8	427.27	54.4	45.4	4	5	0.5	11.0
Faces	Control	9	426.82	54.4	45.4	4	7	0.7	11.9
Faces	Control	10	425.91	54.5	45.4	3	6	0.6	11.5
Faces	Control	11	429.55	54.5	45.4	3	7	0.7	11.9
Faces	Control	12	435.45	54.5	45.4	3	8	0.8	12.4
Faces	Control	13	443.18	54.5	45.5	3	7	0.7	11.9

Appendix Table 14A (Continued). Physical data collected on project horses.

Horse	Group	Week	BW (kg)	Forearm (cm)	Gaskin (cm)	Rib Fat (mm)	Rump Fat (mm)	Rump Fat (cm)	Body Fat (%)
Loreal	Control	0	442.73	53.7	46.6	6	3	0.3	10.1
Loreal	Control	1	450.00	53.7	46.1	6	5	0.5	11.0
Loreal	Control	2	438.64	52.5	46.1	4	7	0.7	11.9
Loreal	Control	3	445.45	52.9	46.3	3	6	0.6	11.5
Loreal	Control	4	447.73	52.9	46.3	3	5	0.5	11.0
Loreal	Control	5	445.00	52.9	46.3	2	4	0.4	10.5
Loreal	Control	6	440.91	52.9	46.3	2	5	0.5	11.0
Loreal	Control	7	445.00	53.0	46.3	2	5	0.5	11.0
Loreal	Control	8	445.45	53.0	46.3	3	5	0.5	11.0
Loreal	Control	9	445.45	53.0	46.3	4	4	0.4	10.5
Loreal	Control	10	454.54	53.0	46.3	3	5	0.5	11.0
Loreal	Control	11	447.73	53.0	46.3	2	9	0.9	12.9
Loreal	Control	12	449.09	53.0	46.3	3	7	0.7	11.9
Loreal	Control	13	451.82	53.0	46.3	3	7	0.7	11.9
Slimer	Control	0	430.00	51.0	45.5	5	4	0.4	10.5
Slimer	Control	1	436.36	49.5	44.6	5	4	0.4	10.5
Slimer	Control	2	436.36	50.3	44.8	3	3	0.3	10.1
Slimer	Control	3	447.73	50.3	45.0	3	3	0.3	10.1
Slimer	Control	4	443.18	50.3	45.0	4	4	0.4	10.5
Slimer	Control	5	435.45	50.4	45.0	4	4	0.4	10.5
Slimer	Control	6	440.45	50.5	45.0	4	4	0.4	10.5
Slimer	Control	7	446.36	50.5	45.0	4	4	0.4	10.5
Slimer	Control	8	449.55	50.5	45.0	3	6	0.6	11.5
Slimer	Control	9	452.73	50.5	45.1	3	11	1.1	13.8
Slimer	Control	10	451.36	50.5	45.1	3	10	1	13.3
Slimer	Control	11	452.27	50.5	45.1	3	11	1.1	13.8
Slimer	Control	12	450.45	50.5	45.1	3	11	1.1	13.8
Slimer	Control	13	452.73	50.5	45.2	3	10	1	13.3

Appendix Table 14A (Continued). Physical data collected on project horses.

Horse	Group	Week	BW (kg)	Forearm (cm)	Gaskin (cm)	Rib Fat (mm)	Rump Fat (mm)	Rump Fat (cm)	Body Fat (%)
Lena	Control	0	432.73	54.5	44.9	4	5	0.5	11.0
Lena	Control	1	442.27	54.3	44.8	2	6	0.6	11.5
Lena	Control	2	442.27	54.5	44.8	4	6	0.6	11.5
Lena	Control	3	447.73	54.5	44.8	3	7	0.7	11.9
Lena	Control	4	442.27	54.5	44.8	3	7	0.7	11.9
Lena	Control	5	440.45	54.5	44.8	3	8	0.8	12.4
Lena	Control	6	436.82	54.4	44.8	2	8	0.8	12.4
Lena	Control	7	445.45	54.6	44.8	2	9	0.9	12.9
Lena	Control	8	452.73	54.6	44.8	2	9	0.9	12.9
Lena	Control	9	454.54	54.6	44.8	2	9	0.9	12.9
Lena	Control	10	447.73	54.6	44.9	2	10	1	13.3
Lena	Control	11	450.45	54.6	45.0	2	10	1	13.3
Lena	Control	12	455.45	54.6	45.0	3	9	0.9	12.9
Lena	Control	13	453.64	54.6	45.0	3	10	1	13.3

Appendix Table 15A. Reproductive ultrasonographic measurements in project horses.

Horse	Group	Cycle	Time to Ovulation (d)	Ovulatory Size (mm)
Dee	RAC	1	45	44
Dee	RAC	2	21	34
Dee	RAC	3	28	37
Freckles	RAC	1	17	37
Freckles	RAC	2	21	40
Freckles	RAC	3	18	38
Freckles	RAC	4	21	33
Freckles	RAC	5	21	37
Missy	RAC	1	24	44
Missy	RAC	2	21	48
Missy	RAC	3	21	38
Mary Kay	RAC	1	18	39
Mary Kay	RAC	2	24	36
Mary Kay	RAC	3	18	43
Mary Kay	RAC	4	17	35
Mary Kay	RAC	5	21	41
Blue	RAC	1	67	35
Scoot	RAC	1	46	30
Scoot	RAC	2	21	39
Scoot	RAC	3	18	30
Prissy	RAC	1	17	30
Prissy	RAC	2	25	36
Prissy	RAC	3	28	28

Appendix Table 15A (Continued). Reproductive ultrasonographic measurements in project horses.

Horse	Group	Cycle	Time to Ovulation (d)	Ovulatory Size (mm)
Sun	Control	1	31	33
Sun	Control	2	46	36
Nina	Control	1	17	35
Nina	Control	2	21	37
Nina	Control	3	21	33
Nina	Control	4	21	44
Marla	Control	1	42	40
Marla	Control	2	18	38
Marla	Control	3	24	35
Faces	Control	1	21	34
Faces	Control	2	31	30
Faces	Control	3	18	38
Loreal	Control	1	10	39
Loreal	Control	2	21	39
Loreal	Control	3	18	36
Loreal	Control	4	24	37
Loreal	Control	5	18	31
Slimer	Control	1	28	33
Slimer	Control	2	21	20
Slimer	Control	3	24	33
Slimer	Control	4	18	35
Lena	Control	1	21	35
Lena	Control	2	25	43
Lena	Control	3	21	35

Appendix Table 16A. Serum leptin concentrations from project horses.

Horse	Group	Week	Leptin (ng/ml)	Leptin Norm.
Dee	RAC	0	1.0709	0.0000
Dee	RAC	1	0.9921	-0.0788
Dee	RAC	2	1.0507	-0.0202
Dee	RAC	3	1.1106	0.0397
Dee	RAC	4	2.3652	1.2943
Dee	RAC	5	1.5354	0.4645
Dee	RAC	6	1.8650	0.7941
Dee	RAC	7	1.4938	0.4229
Dee	RAC	8	1.3546	0.2837
Dee	RAC	9	1.4727	0.4018
Dee	RAC	10	1.6409	0.5700
Dee	RAC	11	2.9632	1.8923
Dee	RAC	12	3.0391	1.9682
Dee	RAC	13	1.4053	0.3344
Missy	RAC	0	1.6626	0.0000
Missy	RAC	1	1.8415	0.1789
Missy	RAC	2	2.0583	0.3957
Missy	RAC	3	1.7053	0.0427
Missy	RAC	4	1.7679	0.1053
Missy	RAC	5	1.5785	-0.0841
Missy	RAC	6	1.9275	0.2649
Missy	RAC	7	1.3984	-0.2642
Missy	RAC	8	1.5913	-0.0713
Missy	RAC	9	1.6041	-0.0585
Missy	RAC	10	1.9138	0.2512
Missy	RAC	11	1.7727	0.1101
Missy	RAC	12	1.9887	0.3261
Missy	RAC	13	2.6180	0.9554

Appendix Table 16A (Continued). Serum leptin concentrations from project horses.

Horse	Group	Week	Leptin (ng/ml)	Leptin Norm.
Mary Kay	RAC	0	1.1724	0.0000
Mary Kay	RAC	1	1.3437	0.1713
Mary Kay	RAC	2	1.5754	0.4030
Mary Kay	RAC	3	1.5782	0.4058
Mary Kay	RAC	4	1.5732	0.4008
Mary Kay	RAC	5	2.0189	0.8465
Mary Kay	RAC	6	2.5116	1.3392
Mary Kay	RAC	7	1.2747	0.1023
Mary Kay	RAC	8	1.6259	0.4535
Mary Kay	RAC	9	2.8423	1.6699
Mary Kay	RAC	10	1.5944	0.4220
Mary Kay	RAC	11	1.5189	0.3465
Mary Kay	RAC	12	1.4649	0.2925
Mary Kay	RAC	13	1.4650	0.2926
Blue	RAC	0	1.4152	0.0000
Blue	RAC	1	1.6653	0.2501
Blue	RAC	2	1.7812	0.3660
Blue	RAC	3	1.3711	-0.0441
Blue	RAC	4	1.7315	0.3163
Blue	RAC	5	1.6740	0.2588
Blue	RAC	6	1.5064	0.0912
Blue	RAC	7	1.4794	0.0642
Blue	RAC	8	1.3684	-0.0468
Blue	RAC	9	1.5455	0.1303
Blue	RAC	10	1.4525	0.0373
Blue	RAC	11	1.4016	-0.0136
Blue	RAC	12	1.4414	0.0262
Blue	RAC	13	1.9265	0.5113

Appendix Table 16A (Continued). Serum leptin concentrations from project horses.

Horse	Group	Week	Leptin (ng/ml)	Leptin Norm.
Scoot	RAC	0	1.9031	0.0000
Scoot	RAC	1	1.6935	-0.2096
Scoot	RAC	2	1.6686	-0.2345
Scoot	RAC	3	1.7425	-0.1606
Scoot	RAC	4	1.0062	-0.8969
Scoot	RAC	5	1.3751	-0.5280
Scoot	RAC	6	1.2248	-0.6783
Scoot	RAC	7	1.0589	-0.8442
Scoot	RAC	8	1.7670	-0.1361
Scoot	RAC	9	1.1917	-0.7114
Scoot	RAC	10	1.2044	-0.6987
Scoot	RAC	11	1.1367	-0.7664
Scoot	RAC	12	1.7414	-0.1617
Scoot	RAC	13	1.2380	-0.6651
Prissy	RAC	0	2.5462	0.0000
Prissy	RAC	1	4.0372	1.4910
Prissy	RAC	2	3.4017	0.8555
Prissy	RAC	3	2.9063	0.3601
Prissy	RAC	4	3.5298	0.9836
Prissy	RAC	5	3.0448	0.4986
Prissy	RAC	6	3.5778	1.0316
Prissy	RAC	7	3.8389	1.2927
Prissy	RAC	8	4.1362	1.5900
Prissy	RAC	9	3.9537	1.4075
Prissy	RAC	10	4.0822	1.5360
Prissy	RAC	11	4.2799	1.7337
Prissy	RAC	12	4.0943	1.5481
Prissy	RAC	13	3.6617	1.1155

Appendix Table 16A (Continued). Serum leptin concentrations from project horses.

Horse	Group	Week	Leptin (ng/ml)	Leptin Norm.
Sun	Control	0	6.2769	0.0000
Sun	Control	1	7.3118	1.0349
Sun	Control	2	6.6174	0.3405
Sun	Control	3	6.6909	0.4140
Sun	Control	4	6.6227	0.3458
Sun	Control	5	7.2456	0.9687
Sun	Control	6	7.3698	1.0929
Sun	Control	7	7.0390	0.7621
Sun	Control	8	7.4070	1.1301
Sun	Control	9	7.5089	1.2320
Sun	Control	10	7.7734	1.4965
Sun	Control	11	8.2132	1.9363
Sun	Control	12	8.0114	1.7345
Sun	Control	13	7.6862	1.4093
Nina	Control	0	3.5952	0.0000
Nina	Control	1	3.8576	0.2624
Nina	Control	2	2.8610	-0.7342
Nina	Control	3	3.6594	0.0642
Nina	Control	4	3.5383	-0.0569
Nina	Control	5	3.3861	-0.2091
Nina	Control	6	3.6747	0.0795
Nina	Control	7	4.0120	0.4168
Nina	Control	8	4.3453	0.7501
Nina	Control	9	4.2011	0.6059
Nina	Control	10	4.3924	0.7972
Nina	Control	11	3.8701	0.2749
Nina	Control	12	4.4252	0.8300
Nina	Control	13	4.2584	0.6632

Appendix Table 16A (Continued). Serum leptin concentrations from project horses.

Horse	Group	Week	Leptin (ng/ml)	Leptin Norm.
Marla	Control	0	2.8376	0.0000
Marla	Control	1	3.6587	0.8211
Marla	Control	2	3.1769	0.3393
Marla	Control	3	2.4040	-0.4336
Marla	Control	4	3.1502	0.3126
Marla	Control	5	3.0988	0.2612
Marla	Control	6	3.0924	0.2548
Marla	Control	7	3.6662	0.8286
Marla	Control	8	2.1348	-0.7028
Marla	Control	9	3.2288	0.3912
Marla	Control	10	2.1701	-0.6675
Marla	Control	11	2.4251	-0.4125
Marla	Control	12	1.9636	-0.8740
Marla	Control	13	1.8144	-1.0232
Faces	Control	0	1.5420	0.0000
Faces	Control	1	1.0542	-0.4878
Faces	Control	2	1.8646	0.3226
Faces	Control	3	1.2614	-0.2806
Faces	Control	4	1.7261	0.1841
Faces	Control	5	1.8479	0.3059
Faces	Control	6	1.5001	-0.0419
Faces	Control	7	1.2459	-0.2961
Faces	Control	8	1.0387	-0.5033
Faces	Control	9	1.0571	-0.4849
Faces	Control	10	1.7649	0.2229
Faces	Control	11	1.6167	0.0747
Faces	Control	12	1.5482	0.0062
Faces	Control	13	1.0369	-0.5051

Appendix Table 16A (Continued). Serum leptin concentrations from project horses.

Horse	Group	Week	Leptin (ng/ml)	Leptin Norm.
Loreal	Control	0	1.5887	0.0000
Loreal	Control	1	1.0860	-0.5027
Loreal	Control	2	1.4758	-0.1129
Loreal	Control	3	1.5048	-0.0839
Loreal	Control	4	1.2146	-0.3741
Loreal	Control	5	1.4498	-0.1389
Loreal	Control	6	1.3194	-0.2693
Loreal	Control	7	1.1254	-0.4633
Loreal	Control	8	1.5935	0.0048
Loreal	Control	9	1.1587	-0.4300
Loreal	Control	10	0.9309	-0.6578
Loreal	Control	11	1.0393	-0.5494
Loreal	Control	12	1.0782	-0.5105
Loreal	Control	13	1.2059	-0.3828
Slimer	Control	0	1.3104	0.0000
Slimer	Control	1	3.9064	2.5960
Slimer	Control	2	1.7764	0.4660
Slimer	Control	3	1.7273	0.4169
Slimer	Control	4	1.8350	0.5246
Slimer	Control	5	1.8128	0.5024
Slimer	Control	6	2.0512	0.7408
Slimer	Control	7	1.9534	0.6430
Slimer	Control	8	2.6900	1.3796
Slimer	Control	9	2.2965	0.9861
Slimer	Control	10	2.2717	0.9613
Slimer	Control	11	2.0993	0.7889
Slimer	Control	12	2.3238	1.0134
Slimer	Control	13	1.4757	0.1653

Appendix Table 16A (Continued). Serum leptin concentrations from project horses.

Horse	Group	Week	Leptin (ng/ml)	Leptin Norm.
Lena	Control	0	1.5564	0.0000
Lena	Control	1	1.5353	-0.0211
Lena	Control	2	1.5676	0.0112
Lena	Control	3	1.3092	-0.2472
Lena	Control	4	1.5996	0.0432
Lena	Control	5	1.5636	0.0072
Lena	Control	6	1.6883	0.1319
Lena	Control	7	1.8818	0.3254
Lena	Control	8	1.7476	0.1912
Lena	Control	9	1.9948	0.4384
Lena	Control	10	2.0381	0.4817
Lena	Control	11	1.8493	0.2929
Lena	Control	12	2.8869	1.3305
Lena	Control	13	2.5553	0.9989

Appendix Table 17A. Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Dee	RAC	0	0.4
Dee	RAC	2	0.4
Dee	RAC	4	0.3
Dee	RAC	6	0.9
Dee	RAC	8	1.5
Dee	RAC	10	1.5
Dee	RAC	12	1.8
Dee	RAC	14	9.0
Dee	RAC	16	2.1
Dee	RAC	18	0.5
Dee	RAC	20	0.3
Dee	RAC	22	0.3
Dee	RAC	24	0.4
Dee	RAC	26	0.3
Dee	RAC	28	0.3
Dee	RAC	30	0.5
Dee	RAC	32	0.8
Dee	RAC	34	2.6
Dee	RAC	36	3.2
Dee	RAC	38	2.5
Dee	RAC	40	1.2
Dee	RAC	42	0.7
Dee	RAC	44	0.4
Dee	RAC	46	0.3
Dee	RAC	48	0.3
Dee	RAC	50	0.4
Dee	RAC	52	0.8
Dee	RAC	54	0.9
Dee	RAC	56	1.2
Dee	RAC	58	1.5
Dee	RAC	60	1.6
Dee	RAC	62	0.7
Dee	RAC	64	0.2
Dee	RAC	66	0.2
Dee	RAC	68	0.3
Dee	RAC	70	0.3
Dee	RAC	72	0.3
Dee	RAC	74	0.3
Dee	RAC	76	0.4
Dee	RAC	78	1.1
Dee	RAC	80	1.3
Dee	RAC	82	1.2

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Dee	RAC	84	1.2
Dee	RAC	86	2.2
Dee	RAC	88	2.8
Dee	RAC	90	0.9
Missy	RAC	0	0.7
Missy	RAC	2	0.4
Missy	RAC	4	0.7
Missy	RAC	6	0.6
Missy	RAC	8	0.6
Missy	RAC	10	0.3
Missy	RAC	12	0.7
Missy	RAC	14	1.1
Missy	RAC	16	1.5
Missy	RAC	18	1.6
Missy	RAC	20	0.3
Missy	RAC	22	0.7
Missy	RAC	24	0.4
Missy	RAC	26	0.5
Missy	RAC	28	0.3
Missy	RAC	30	0.5
Missy	RAC	32	0.3
Missy	RAC	34	0.6
Missy	RAC	36	0.6
Missy	RAC	38	0.7
Missy	RAC	40	0.9
Missy	RAC	42	0.6
Missy	RAC	44	0.6
Missy	RAC	46	0.4
Missy	RAC	48	0.4
Missy	RAC	50	0.5
Missy	RAC	52	0.5
Missy	RAC	54	0.4
Missy	RAC	56	0.4
Missy	RAC	58	0.4
Missy	RAC	60	0.5
Missy	RAC	62	0.7
Missy	RAC	64	0.6
Missy	RAC	66	0.3
Missy	RAC	68	1.1
Missy	RAC	70	0.4
Missy	RAC	72	0.4

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Missy	RAC	74	0.1
Missy	RAC	76	0.3
Missy	RAC	78	0.3
Missy	RAC	80	0.3
Missy	RAC	82	0.5
Missy	RAC	84	0.6
Missy	RAC	86	0.7
Missy	RAC	88	0.8
Missy	RAC	90	0.6
Mary Kay	RAC	0	0.4
Mary Kay	RAC	2	0.4
Mary Kay	RAC	4	0.6
Mary Kay	RAC	6	1.2
Mary Kay	RAC	8	0.6
Mary Kay	RAC	10	0.4
Mary Kay	RAC	12	0.2
Mary Kay	RAC	14	0.4
Mary Kay	RAC	16	0.4
Mary Kay	RAC	18	0.4
Mary Kay	RAC	20	0.3
Mary Kay	RAC	22	1.4
Mary Kay	RAC	24	1.4
Mary Kay	RAC	26	1.3
Mary Kay	RAC	28	0.8
Mary Kay	RAC	30	0.3
Mary Kay	RAC	32	0.4
Mary Kay	RAC	34	0.4
Mary Kay	RAC	36	0.2
Mary Kay	RAC	38	0.3
Mary Kay	RAC	40	0.2
Mary Kay	RAC	42	1.3
Mary Kay	RAC	44	0.8
Mary Kay	RAC	46	1.0
Mary Kay	RAC	48	1.1
Mary Kay	RAC	50	1.1
Mary Kay	RAC	52	0.5
Mary Kay	RAC	54	1.2
Mary Kay	RAC	56	0.2
Mary Kay	RAC	58	0.5
Mary Kay	RAC	60	0.3
Mary Kay	RAC	62	0.4

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Mary Kay	RAC	64	1.6
Mary Kay	RAC	66	1.5
Mary Kay	RAC	68	0.5
Mary Kay	RAC	70	0.6
Mary Kay	RAC	72	3.2
Mary Kay	RAC	74	0.4
Mary Kay	RAC	76	0.3
Mary Kay	RAC	78	0.3
Mary Kay	RAC	80	0.6
Mary Kay	RAC	82	0.9
Mary Kay	RAC	84	3.1
Mary Kay	RAC	86	0.6
Mary Kay	RAC	88	1.7
Mary Kay	RAC	90	0.6
Blue	RAC	0	1.1
Blue	RAC	2	1.8
Blue	RAC	4	3.0
Blue	RAC	6	14.2
Blue	RAC	8	17.1
Blue	RAC	10	6.6
Blue	RAC	12	2.3
Blue	RAC	14	0.9
Blue	RAC	16	0.8
Blue	RAC	18	0.9
Blue	RAC	20	0.5
Blue	RAC	22	1.1
Blue	RAC	24	2.0
Blue	RAC	26	6.8
Blue	RAC	28	6.7
Blue	RAC	30	5.1
Blue	RAC	32	2.1
Blue	RAC	34	0.7
Blue	RAC	36	0.9
Blue	RAC	38	0.8
Blue	RAC	40	0.7
Blue	RAC	42	1.1
Blue	RAC	44	1.3
Blue	RAC	46	0.6
Blue	RAC	48	1.0
Blue	RAC	50	0.7
Blue	RAC	52	0.8

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Blue	RAC	54	0.8
Blue	RAC	56	0.6
Blue	RAC	58	0.4
Blue	RAC	60	0.5
Blue	RAC	62	0.3
Blue	RAC	64	1.1
Blue	RAC	66	0.9
Blue	RAC	68	0.7
Blue	RAC	70	0.7
Blue	RAC	72	0.7
Blue	RAC	74	0.5
Blue	RAC	76	0.6
Blue	RAC	78	0.6
Blue	RAC	80	0.7
Blue	RAC	82	0.8
Blue	RAC	84	0.4
Blue	RAC	86	1.0
Blue	RAC	88	0.7
Blue	RAC	90	0.3
Scoot	RAC	0	2.0
Scoot	RAC	2	0.8
Scoot	RAC	4	0.5
Scoot	RAC	6	0.7
Scoot	RAC	8	0.9
Scoot	RAC	10	0.3
Scoot	RAC	12	0.3
Scoot	RAC	14	0.3
Scoot	RAC	16	0.2
Scoot	RAC	18	0.1
Scoot	RAC	20	0.3
Scoot	RAC	22	0.1
Scoot	RAC	24	0.1
Scoot	RAC	26	0.1
Scoot	RAC	28	0.4
Scoot	RAC	30	0.3
Scoot	RAC	32	0.2
Scoot	RAC	34	0.1
Scoot	RAC	36	0.1
Scoot	RAC	38	0.4
Scoot	RAC	40	0.1
Scoot	RAC	42	0.2

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Scoot	RAC	44	0.2
Scoot	RAC	46	0.1
Scoot	RAC	48	0.2
Scoot	RAC	50	0.1
Scoot	RAC	52	0.4
Scoot	RAC	54	0.4
Scoot	RAC	56	0.1
Scoot	RAC	58	0.2
Scoot	RAC	60	0.3
Scoot	RAC	62	0.2
Scoot	RAC	64	0.8
Scoot	RAC	66	2.0
Scoot	RAC	68	0.2
Scoot	RAC	70	0.1
Scoot	RAC	72	0.1
Scoot	RAC	74	0.6
Scoot	RAC	76	0.1
Scoot	RAC	78	0.6
Scoot	RAC	80	0.5
Scoot	RAC	82	0.4
Scoot	RAC	84	0.1
Scoot	RAC	86	0.1
Scoot	RAC	88	0.1
Scoot	RAC	90	0.1
Prissy	RAC	0	0.7
Prissy	RAC	2	0.3
Prissy	RAC	4	0.4
Prissy	RAC	6	1.0
Prissy	RAC	8	2.1
Prissy	RAC	10	0.5
Prissy	RAC	12	0.6
Prissy	RAC	14	1.0
Prissy	RAC	16	0.9
Prissy	RAC	18	0.7
Prissy	RAC	20	0.4
Prissy	RAC	22	0.6
Prissy	RAC	24	0.6
Prissy	RAC	26	0.5
Prissy	RAC	28	0.5
Prissy	RAC	30	0.4
Prissy	RAC	32	0.3

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Prissy	RAC	34	0.4
Prissy	RAC	36	0.4
Prissy	RAC	38	0.3
Prissy	RAC	40	0.2
Prissy	RAC	42	0.2
Prissy	RAC	44	0.3
Prissy	RAC	46	0.3
Prissy	RAC	48	0.2
Prissy	RAC	50	0.2
Prissy	RAC	52	0.2
Prissy	RAC	54	0.2
Prissy	RAC	56	0.2
Prissy	RAC	58	0.2
Prissy	RAC	60	0.2
Prissy	RAC	62	0.2
Prissy	RAC	64	0.2
Prissy	RAC	66	0.2
Prissy	RAC	68	0.2
Prissy	RAC	70	0.2
Prissy	RAC	72	0.2
Prissy	RAC	74	0.2
Prissy	RAC	76	0.1
Prissy	RAC	78	0.2
Prissy	RAC	80	0.2
Prissy	RAC	82	0.2
Prissy	RAC	84	0.3
Prissy	RAC	86	0.2
Prissy	RAC	88	0.2
Prissy	RAC	90	0.2
Sun	Control	0	0.8
Sun	Control	2	0.5
Sun	Control	4	1.4
Sun	Control	6	3.0
Sun	Control	8	3.5
Sun	Control	10	2.5
Sun	Control	12	2.4
Sun	Control	14	2.5
Sun	Control	16	3.2
Sun	Control	18	5.6
Sun	Control	20	2.3
Sun	Control	22	1.3

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Sun	Control	24	1.3
Sun	Control	26	1.2
Sun	Control	28	0.7
Sun	Control	30	0.6
Sun	Control	32	0.4
Sun	Control	34	0.5
Sun	Control	36	0.4
Sun	Control	38	0.3
Sun	Control	40	0.3
Sun	Control	42	0.4
Sun	Control	44	0.4
Sun	Control	46	0.5
Sun	Control	48	0.2
Sun	Control	50	0.2
Sun	Control	52	0.4
Sun	Control	54	0.3
Sun	Control	56	0.3
Sun	Control	58	0.3
Sun	Control	60	0.3
Sun	Control	62	0.3
Sun	Control	64	0.4
Sun	Control	66	0.3
Sun	Control	68	0.3
Sun	Control	70	0.7
Sun	Control	72	0.3
Sun	Control	74	0.7
Sun	Control	76	0.5
Sun	Control	78	0.4
Sun	Control	80	0.5
Sun	Control	82	0.3
Sun	Control	84	0.2
Sun	Control	86	0.3
Sun	Control	88	0.2
Sun	Control	90	0.5
Nina	Control	0	0.9
Nina	Control	2	1.1
Nina	Control	4	1.0
Nina	Control	6	2.9
Nina	Control	8	3.7
Nina	Control	10	12.1
Nina	Control	12	5.8

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Nina	Control	14	2.7
Nina	Control	16	1.4
Nina	Control	18	1.6
Nina	Control	20	1.2
Nina	Control	22	1.2
Nina	Control	24	1.4
Nina	Control	26	2.7
Nina	Control	28	3.3
Nina	Control	30	4.8
Nina	Control	32	2.5
Nina	Control	34	3.7
Nina	Control	36	2.0
Nina	Control	38	1.4
Nina	Control	40	1.3
Nina	Control	42	1.3
Nina	Control	44	1.2
Nina	Control	46	1.3
Nina	Control	48	2.2
Nina	Control	50	2.3
Nina	Control	52	5.3
Nina	Control	54	6.8
Nina	Control	56	8.4
Nina	Control	58	2.5
Nina	Control	60	1.3
Nina	Control	62	1.2
Nina	Control	64	0.8
Nina	Control	66	1.0
Nina	Control	68	1.4
Nina	Control	70	3.0
Nina	Control	72	4.0
Nina	Control	74	5.3
Nina	Control	76	4.8
Nina	Control	78	2.8
Nina	Control	80	3.0
Nina	Control	82	1.6
Nina	Control	84	1.5
Nina	Control	86	1.6
Nina	Control	88	1.8
Nina	Control	90	1.8
Marla	Control	0	2.2
Marla	Control	2	2.0

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Marla	Control	4	1.1
Marla	Control	6	1.0
Marla	Control	8	1.5
Marla	Control	10	2.0
Marla	Control	12	1.1
Marla	Control	14	1.5
Marla	Control	16	1.3
Marla	Control	18	1.7
Marla	Control	20	1.7
Marla	Control	22	1.8
Marla	Control	24	2.0
Marla	Control	26	1.0
Marla	Control	28	1.0
Marla	Control	30	1.4
Marla	Control	32	1.3
Marla	Control	34	1.1
Marla	Control	36	1.5
Marla	Control	38	1.5
Marla	Control	40	1.7
Marla	Control	42	1.1
Marla	Control	44	1.1
Marla	Control	46	1.7
Marla	Control	48	2.0
Marla	Control	50	2.2
Marla	Control	52	7.2
Marla	Control	54	9.3
Marla	Control	56	3.9
Marla	Control	58	3.7
Marla	Control	60	2.4
Marla	Control	62	2.6
Marla	Control	64	1.3
Marla	Control	66	1.9
Marla	Control	68	2.4
Marla	Control	70	1.2
Marla	Control	72	1.4
Marla	Control	74	2.4
Marla	Control	76	3.4
Marla	Control	78	4.5
Marla	Control	80	6.3
Marla	Control	82	2.2
Marla	Control	84	1.9
Marla	Control	86	1.5

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Marla	Control	88	1.4
Marla	Control	90	1.0
Faces	Control	0	1.8
Faces	Control	2	5.0
Faces	Control	4	3.4
Faces	Control	6	2.4
Faces	Control	8	2.3
Faces	Control	10	2.9
Faces	Control	12	1.5
Faces	Control	14	1.2
Faces	Control	16	1.6
Faces	Control	18	2.1
Faces	Control	20	2.3
Faces	Control	22	3.7
Faces	Control	24	2.0
Faces	Control	26	6.6
Faces	Control	28	7.0
Faces	Control	30	1.0
Faces	Control	32	2.2
Faces	Control	34	1.9
Faces	Control	36	1.7
Faces	Control	38	1.6
Faces	Control	40	1.1
Faces	Control	42	1.9
Faces	Control	44	3.4
Faces	Control	46	2.1
Faces	Control	48	2.7
Faces	Control	50	4.7
Faces	Control	52	9.5
Faces	Control	54	2.8
Faces	Control	56	3.2
Faces	Control	58	2.1
Faces	Control	60	1.7
Faces	Control	62	1.3
Faces	Control	64	1.6
Faces	Control	66	1.6
Faces	Control	68	1.0
Faces	Control	70	1.2
Faces	Control	72	1.3
Faces	Control	74	1.7
Faces	Control	76	2.5

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Faces	Control	78	1.4
Faces	Control	80	1.0
Faces	Control	82	1.4
Faces	Control	84	0.9
Faces	Control	86	1.7
Faces	Control	88	0.6
Faces	Control	90	0.5
Loreal	Control	0	0.4
Loreal	Control	2	0.2
Loreal	Control	4	0.6
Loreal	Control	6	1.0
Loreal	Control	8	2.3
Loreal	Control	10	3.3
Loreal	Control	12	2.0
Loreal	Control	14	0.4
Loreal	Control	16	0.5
Loreal	Control	18	0.3
Loreal	Control	20	0.1
Loreal	Control	22	0.5
Loreal	Control	24	0.4
Loreal	Control	26	1.1
Loreal	Control	28	3.1
Loreal	Control	30	5.0
Loreal	Control	32	2.4
Loreal	Control	34	1.0
Loreal	Control	36	0.9
Loreal	Control	38	0.3
Loreal	Control	40	0.2
Loreal	Control	42	0.3
Loreal	Control	44	0.6
Loreal	Control	46	2.2
Loreal	Control	48	4.0
Loreal	Control	50	3.2
Loreal	Control	52	2.0
Loreal	Control	54	1.0
Loreal	Control	56	0.2
Loreal	Control	58	0.3
Loreal	Control	60	0.1
Loreal	Control	62	0.4
Loreal	Control	64	1.2
Loreal	Control	66	1.8

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Loreal	Control	68	1.0
Loreal	Control	70	0.3
Loreal	Control	72	0.6
Loreal	Control	74	0.2
Loreal	Control	76	0.1
Loreal	Control	78	0.6
Loreal	Control	80	0.1
Loreal	Control	82	0.1
Loreal	Control	84	0.5
Loreal	Control	86	0.6
Loreal	Control	88	1.5
Loreal	Control	90	3.8
Slimer	Control	0	1.8
Slimer	Control	2	0.7
Slimer	Control	4	0.5
Slimer	Control	6	0.5
Slimer	Control	8	1.0
Slimer	Control	10	1.0
Slimer	Control	12	0.7
Slimer	Control	14	0.5
Slimer	Control	16	0.7
Slimer	Control	18	0.5
Slimer	Control	20	0.3
Slimer	Control	22	0.4
Slimer	Control	24	0.3
Slimer	Control	26	0.2
Slimer	Control	28	0.1
Slimer	Control	30	0.3
Slimer	Control	32	0.3
Slimer	Control	34	0.3
Slimer	Control	36	0.3
Slimer	Control	38	0.2
Slimer	Control	40	0.2
Slimer	Control	42	0.2
Slimer	Control	44	0.2
Slimer	Control	46	0.3
Slimer	Control	48	0.3
Slimer	Control	50	0.2
Slimer	Control	52	0.2
Slimer	Control	54	0.4
Slimer	Control	56	0.2

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Slimer	Control	58	0.2
Slimer	Control	60	0.2
Slimer	Control	62	0.2
Slimer	Control	64	0.1
Slimer	Control	66	0.1
Slimer	Control	68	0.3
Slimer	Control	70	0.3
Slimer	Control	72	0.1
Slimer	Control	74	0.1
Slimer	Control	76	0.2
Slimer	Control	78	0.2
Slimer	Control	80	0.5
Slimer	Control	82	0.5
Slimer	Control	84	0.6
Slimer	Control	86	0.7
Slimer	Control	88	1.4
Slimer	Control	90	0.8
Lena	Control	0	0.9
Lena	Control	2	0.6
Lena	Control	4	0.8
Lena	Control	6	1.2
Lena	Control	8	2.0
Lena	Control	10	3.6
Lena	Control	12	2.2
Lena	Control	14	0.5
Lena	Control	16	0.6
Lena	Control	18	0.5
Lena	Control	20	0.5
Lena	Control	22	0.4
Lena	Control	24	0.2
Lena	Control	26	0.5
Lena	Control	28	1.2
Lena	Control	30	3.0
Lena	Control	32	3.9
Lena	Control	34	1.2
Lena	Control	36	0.4
Lena	Control	38	0.2
Lena	Control	40	0.3
Lena	Control	42	0.2
Lena	Control	44	0.3
Lena	Control	46	0.4

Appendix Table 17A (Continued). Serum LH concentrations from project horses.

Horse	Group	Day	LH (ng/ml)
Lena	Control	48	0.5
Lena	Control	50	0.6
Lena	Control	52	0.8
Lena	Control	54	1.1
Lena	Control	56	1.6
Lena	Control	58	0.5
Lena	Control	60	0.2
Lena	Control	62	0.2
Lena	Control	64	0.2
Lena	Control	66	0.6
Lena	Control	68	0.1
Lena	Control	70	0.1
Lena	Control	72	0.2
Lena	Control	74	0.5
Lena	Control	76	0.4
Lena	Control	78	0.6
Lena	Control	80	0.9
Lena	Control	82	2.0
Lena	Control	84	0.7
Lena	Control	86	0.1
Lena	Control	88	0.3
Lena	Control	90	0.3

Appendix Table 18A. Mean LH concentrations by group for project horses.

Group	Day	LH (ng/ml)
RAC	0	0.88
RAC	2	0.68
RAC	4	0.92
RAC	6	3.10
RAC	8	3.80
RAC	10	1.60
RAC	12	0.98
RAC	14	2.12
RAC	16	0.98
RAC	18	0.70
RAC	20	0.35
RAC	22	0.70
RAC	24	0.81
RAC	26	1.58
RAC	28	1.50
RAC	30	1.18
RAC	32	0.68
RAC	34	0.80
RAC	36	0.90
RAC	38	0.83
RAC	40	0.55
RAC	42	0.68
RAC	44	0.60
RAC	46	0.45
RAC	48	0.53
RAC	50	0.50
RAC	52	0.53
RAC	54	0.65
RAC	56	0.45
RAC	58	0.53
RAC	60	0.57
RAC	62	0.42
RAC	64	0.75
RAC	66	0.85
RAC	68	0.50
RAC	70	0.38
RAC	72	0.82
RAC	74	0.35
RAC	76	0.30
RAC	78	0.52
RAC	80	0.60
RAC	82	0.67

Appendix Table 18A (Continued). Mean LH concentrations by group for project horses.

Group	Day	LH (ng/ml)
RAC	84	0.95
RAC	86	0.80
RAC	88	1.05
RAC	90	0.45
Control	0	1.26
Control	2	1.44
Control	4	1.26
Control	6	1.71
Control	8	2.33
Control	10	3.91
Control	12	2.24
Control	14	1.33
Control	16	1.33
Control	18	1.76
Control	20	1.20
Control	22	1.33
Control	24	1.09
Control	26	1.90
Control	28	2.34
Control	30	2.30
Control	32	1.86
Control	34	1.39
Control	36	1.03
Control	38	0.79
Control	40	0.73
Control	42	0.77
Control	44	1.03
Control	46	1.21
Control	48	1.70
Control	50	1.91
Control	52	3.63
Control	54	3.10
Control	56	2.54
Control	58	1.37
Control	60	0.89
Control	62	0.89
Control	64	0.80
Control	66	1.04
Control	68	0.93
Control	70	0.97
Control	72	1.13

Appendix Table 18A (Continued). Mean LH concentrations by group for project horses.

Group	Day	LH (ng/ml)
Control	76	1.70
Control	78	1.50
Control	80	1.76
Control	82	1.16
Control	84	0.90
Control	86	0.93
Control	88	1.03
Control	90	1.24

Appendix Table 19A. AUC values used to calculate total LH concentrations between groups

Horse	Group	AUC
Dee	RAC	103.3
Missy	RAC	52.5
Mary Kay	RAC	71.7
Blue	RAC	186.6
Scoot	RAC	31.0
Prissy	RAC	35.5
Sun	Control	87.8
Nina	Control	254.7
Marla	Control	201.2
Faces	Control	216.8
Loreal	Control	111.1
Slimer	Control	37.0
Lena	Control	75.6

VITA

Russell Derek Kriewald was born August 10, 1983 in Hondo, Texas, where he would reside until graduating with Honors from Hondo High School in May 2001. His background in agriculture and success in 4-H and FFA activities led him to pursue a life of his own in College Station, Texas at Texas A&M University, where he would first begin his course work in August 2001.

During this time, he would become active in the Biomedical Science Association, Texas A&M Horsemen's Association, and the American Collegiate Horsemen's Association, to which he served as President in the latter two organizations. Russell was also a member of the 2002 TAMU Collegiate Horse Judging Team coached by Kris Wilson and Dr. Gary Potter. He served as a student worker at the TAMU Horse Center beginning in 2002, and was promoted to be the Assistant Manager in 2004. In May 2005, he was awarded a Bachelor of Science degree in Animal Science.

Upon graduation, Russell applied to the graduate program at Texas A&M University, and began working towards his Master of Science degree under Dr. Martha Vogelsang. In the spring of 2007, he was hired as a teaching assistant for the Animal Science Department, where he gained experience teaching general animal science courses, and equine behavior and training. Russell completed his career at Texas A&M University and accepted his Master of Science degree in Animal Science in May 2008.

Russell is now pursuing a career at the University of Tennessee-Knoxville, where he is lecturing about equine sciences including horse evaluation, management, behavior and training. Contact: Dept. of Animal Science, 249 Kleberg Center, 2471 TAMU, College Station, TX 77843.